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THE TUBERCULOSIS PROBLEM VIEWED IN THE LIGHT OF RECENT PATHOLOGIC STUDIES*

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IT IS with the greatest of pleasure and enthusiasm that I find myself returning to this platform after six years of absence. I am thankful to my many good friends who were instrumental in this cause. Most of all, I am deeply conscious of the honor bestowed upon me to give this lecture in honor of one of America's great pathologists, and a pioneer teacher of that subject.

Apart from the more enjoyable reactions, however, I feel a sense of inferiority: First, in attempting to do honor to Dr. Bell; and, second, in bringing "tuberculosis to Newcastle."

In the presentation of the subject of the evening, there are two great principles that may well be kept in mind. First, it is important always to be aware of a changing world.

"The old order changeth, yielding place to new," applies in evolution, general science, general medicine, tuberculosis, et cetera, as well as to King Arthur's knights. It is equally important to realize that as a result of this change and the limitations of man's ability, *finality* is an illusion that, like the horizon, is never reached.

With these facts in mind to temper our assertions, let us approach the tuberculosis problem with restraint but with confidence in our special senses and the ability to reason from elementary premises.

As a result of change in man's environment, the infection rate of tuberculosis has also been changing. Dating from antiquity, urbanization and industrialization have caused every race to pass through a marked increase in tuberculosis, roughly following the pathways of civilization. The important features are that they have increased, first, the number of contacts by bringing people closer together, and, second, have imposed more severe working conditions that have enhanced the progression of the disease.

Practically every people has gone through such a cycle from a negligible infection rate in the more primitive, to a high rate in the congested and industrialized city.

After reaching a peak of mortality, however, each group apparently settled back to a constant rate when a balance was set up by natural immunizing forces against the spreading disease. Many who became infected made uneventful recoveries, and with the recovery came a relative increase in resistance that protected against subsequent infections. Had this not happened, the race would have become extinct long ago. This balance kept its status quo down the centuries, until it was upset by the discovery of the tubercle bacillus, which has effectively brought the public health machinery into play against the cause and effects of the disease. The results directly and indirectly seemed to have caused the infection rate to fall and along with it the morbidity and mortality. Today the death rate is only about one-tenth what it was at the peak before 1882, and the infection rate is almost parallel with it.

With these changes and dependent upon them, the infection rate has kept on decreasing as tuberculin surveys over the past fifty years indicate. If the rate continues to decrease, the human race will eventually be infection-free, a

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TABLE I—HYPOTHETICAL SHIFT FROM YOUNGER TO OLDER AGE GROUPS AS THE INFECTION RATE DECREASES

Infection Rate per year	Years	% Childhood	% Adult	Ratio A/Ch	% Uninfected	Ratio I/U
8%-100% by	12.5	100	0	0	0	0
5%-100% by	20	70	30	0.4:1	0	0
3%-100% by	33	42	58	1.4:1	0	0
2%-100% by	50	28	72	2.6:1	0	0
1.5%- 91% by	60	21	70	3.5:1	9%	10:1
1.0%- 60% by	60	14	46	3.3:1	40%	1.5:1
0.5%- 30% by	60	7	23	3.3:1	70%	0.4:1

condition comparable to that existing in America's cattle herds.

As shown in Table I, this state has already been reached in most communities where the infection rate is below 1.5 per cent.

Another result of this change is that as more and more children reach adult life without infection, there is going to be a relative increase in the number of infections in adult life, limited only by the number of years of that age period. It should be pointed out at this time, however, that this infection incidence has no bearing on the relative prevalence of disease in the two age groups, information concerning which I have no accurate data at present.

Such a preliminary view of the shifting sands on which accurate statistics may be compiled should make us realize how difficult it has been in the past and how risky it is now to present anything purporting to be permanent or accurate. The types of infection, ages of host, ages and stages of the disease, and the myriad accidents of life coming to play on any infection, cause this condition to exist. Nevertheless, an attempt will be made to include as many facts as possible in a comprehensive outline.

So long as straight morbid anatomy is concerned, a classification may be made; but this cannot be done if it is to serve all purposes. It must be dovetailed into epidemiology and pathogenicity, as well as clinical and x-ray findings, to be comprehensive.

A feature of major importance, therefore, towards this end seems to be to recognize tuberculosis as an evolutionary process. In the vast amount of study on the disease, little emphasis has been placed upon the *age of any particular process.* True, the clinical groupings of "a, b, and c" for each stage of disease, and the terms fibroid and pneumonic, point in this direction, but it does not cover all phases of the disease.

Like anything else biological, tuberculosis has a beginning, a development, and an ending. The outcome of any case becomes easier to foretell when the "phase" or age of the disease is known. At the beginning or "sunrise" of the disease nobody can tell what will be the extent of involvement, the virulence of the germ, or the resistance of the host. Many patients have only a small lesion that soon becomes fatal; others will heal uneventfully. An overwhelming process may be rapidly fatal or drawn out into a chronic process when fibrosis has walled off cavities and calcification has been well established. After fibrosis has become predominant with the clearing of exudates and the appearance of emphysema, the "sundown" of the disease appears, bringing with it many distressing sequelæ, such as emphysema, bronchiectasis, right heart failure, etc., some of which may not be recognized as tuberculosis at all. This stage is vastly different from the beginning stage.

In order to take more cognizance of the *age of the disease,* I have attempted in recent years to establish criteria of age wherever possible. For primary tubercles the results have been gratifying in the majority of cases. In later stages of the disease it has not been so promising.

The best that can be offered is a classification that takes into consideration the rather short beginning and variable middle and end stages. In submitting such a plan it must be

stressed that it is used only as a skeleton around which the various facts may be arranged. There is no intent in offering a substitute for the present order which is altered from time to time as necessity demands or new facts dictate, by properly constituted committees. Like many other innovations, this new classification is bound to have imperfections. For example, the middle period may represent a long succession of new episodes as the disease spreads. The end may be sudden as a terminal hemorrhage, or may be drawn out over years. But such variations cannot be controlled by any classification, old or new.

The proposed outline considers tuberculosis infection as a closely interwoven entity with predominant trends in first infection on one extreme, and the reinfection on the other, with a blending of the two in the intervening types. It considers first an underlying pathology with the evolutionary development of the process and the resulting clinical and x-ray findings as supplementary to them wherever they make their appearance. A chronological sequence will be adhered to as much as possible. It is to be a living or moving caravan that will keep pace with all the changes.

Pathogenesis and Elementary Pathology of Tuberculous Infection with a Few Dependent Clinical and X-ray Findings

I. *First Infection*—may be at any age. Clinical findings are slight. Usually there is a perifocal infiltration as shown on the x-ray.
(The tuberculin reaction turns positive three weeks to three months after infection; usually the time is shorter as the mass of infection increases.)

A. Childhood—in the past generations they were in the majority. They may be classed on a basis of *quantity* of tissue involved, as:
1. Minimal—with only a few lobules, about 1-2 cm. of inflammatory zone.
2. Moderately Advanced—several lobules, about 5 cm. of inflammatory zone.
3. Far Advanced—about 10 cm. of inflammatory zone.

B. Adult—greatly on the increase as a result of decreasing rate of infection. Some typical, others atypical—prone to simulate reinfection with decreasing size of lymph node complex compared to the childhood type.

C. Typical evolution of all primary lesions.
1. Early evolution—progressive from local bronchopneumonia with focal and perifocal inflammation, caseation, cavity formation, depending on the size of the lesion.
(a) First stage (Ranke) with lymph nodes becoming progressively involved over the first few weeks and months, progresses towards the blood stream. Focal and perifocal as in lungs.
(b) Second stage (Ranke) with generalization in the blood stream, to the lungs and beyond. The foci vary from a few to myriads, may spread to secondary lesions.
2. Middle evolutionary period. The lesion becomes more stationary and focal; lesions of varying stages of regression, with a gradual thickening of capsule.
3. Late evolutionary period—"foreign body" calcareous lesions of stony hardness, tuberculin becoming negative. Some lesions become completely resorbed.

II. *Disease Emanating from Primary Lesions—"Endogenous Reinfection."*

A. Rapid progression from primary.
1. Local spread.
(a) Rupture—open lesion into bronchioles or bronchi with bronchial extension (descending endobronchitis).
(b) Overflow to the blood stream (artery, more commonly veins); and lymphatics.
2. Lymph node episodes, lead to
(a) Perivascular effusion.
(1) Retrograde lymph flow clearing without foci of caseous bronchopneumonia.
(2) Caseous pneumonia with varying degrees of ulceration and fibrosis.
(b) Enlarged nodes in and around the main bronchi and hilum leading to,
(1) Bronchitis (hilum catarrh of Ranke) due to filtering through of the bacilli, toxins, etc., and a desiccation of mucus causing cough, wheezing, etc.
(2) Atelectasis.
a. Without parenchymal disease.
1. Clearing with mild emphysema.
b. With parenchymal disease.
1. A "lobitis" or fibroid type with little caseation, blends into
2. A caseous pneumonia, or bronchopneumonia.
(c) Rupture—bronchial extension to lung parenchyma with acinous nodose, bronchopneumonia and pneumonic lesions; also to pleura, to mediastinum, to esophagus.

B. Exacerbation of a primary—after a long period of quiescence.
1. Local lesion.
(a) Rupture—also daughter colony infiltrates, rupture resulting in bronchial spread, with lesions characteristic of bronchial spread.
2. Lymph node.
(a) Slow flowing into bronchial and vascular routes.
(b) Late rupture with bronchial spread.
3. Hematogenous lesions deposited usually from vascular (pulmonary) extension.
(a) Lungs—forming miliary and nodular seeding that may ulcerate into

thin-walled and "alternative" cavaties. Few symptoms.
(b) General—all forms of extrapulmonary lesions, and generalized miliary tuberculosis.

III. *Exogenous Infections.*
A. Superinfection — on another smouldering process.
B. Reinfection—on a completely healed process, all types of pathology of reinfection.

IV. *Quantity, Character and Position of Reinfection Lesion.*
(They usually begin in the upper and posterior aspects of the lungs. Most commonly in the subapical bronchi in progressive disease; in apical bronchi in benign forms; less in horizontal and apex of lower lobe.)
A. Minimal in quantity.
1. Early age of lesion; tiny flecks on the x-ray picture in second interspace and apex; no extension; if Mantoux is negative then turns positive, it is probably a primary.
2. Middle age of lesion; flecks encapsulated; no extension; Mantoux positive.
3. Late age of lesion.
(a) Flecks becoming encapsulated or healed—also include typical Puhl, Simon Stefko nodular foci.

B. Moderately advanced.
1. Early age period; first few months after onset; infiltrates; flecks; located as in minimal lesions; but involving a larger area. May or may not have clinical symptoms.
2. Middle age period; months to years and decades after infection.
(a) Fibrocaseous, ulcerative, progressive lesions. Late nodular lesions, round infiltrates, acinous nodose tubercles, fibrocaseous and ulcerative lesions, cloudy infiltrates and bronchopneumonia with the process beginning to appear in the anterior bronchi. Spread usually by bronchi, but also by blood and lymph.
(b) Fibroid, healing, healed, cleared with emphysematous residue; retrogressive. Sometimes bullous emphysema in apices.

C. Far advanced.
1. Early age period—first few months after onset—massive infiltrates, pneumonia, etc.
2. Middle age period—from months to years after infection; fibrocaseous, ulcerative and pneumonic—extending to the anterior parts of the lungs.
3. Late age period—months to decades after infection.
(a) Terminal; months to years after infection, all other locations and also gravity infiltrates and cavities—progressive. Spread by bronchi, blood and lymph channels.
(b) Healed, years to decades after infection, healing fibroid — retrogressive, bulbous emphysema in bases.

V. *Extra-Pulmonary Tuberculosis.*
Hematogenous—with generalized miliary spread. Pleurisy—bronchial, tracheal, laryngeal, gastro-intestinal, genito-urinary, bone, skin, meninges, etc.

VI. *Paratuberculosis*—disease phenomena, due to
(1) Filtrable.
(2) Non-acid-fast.
(3) Atypical forms of the tubercle parasite, largely hypothetical.

VII. *Complications and Sequelæ*—Hemorrhage, fatty changes, amyloid, emphysema, bronchiectasis, etc.

VIII. *Associated Diseases.*
Carcinoma, diabetes, syphilis, heart disease, pregnancy, etc.

To clarify this outline, an exposition of most types will be given with a well chosen case history and with pathologic findings for a few of the types not ordinarily understood.

As mentioned before, the first infection in tuberculosis may be at any age. In past generations it has been more common in children, but there is taking place a gradual shift into the adult ages as the infection rate decreases. There is a considerable difference, however, in the appearance of the infection in children and that occurring for the first time in adults.

Classical Primary Lesions

As a result of the classical description of Parrot, Küss, H. Albrecht, E. Albrecht, Ghon, Ranke, Opie, Armand-Delille, Lestocquoy, and others, the primary infection in children is well understood. There is a local infection that progresses towards the blood stream through the lymphatics and ultimately results in a spread throughout the body. But this is about the only thing in common even in children. There are no two infections that appear exactly alike. Some infections occurring in certain colored and aboriginal peoples cause a violent local perifocal reaction that sometimes progresses to a caseous pneumonia. The lymphatics may become tremendously involved and there may be an extensive hematogenous dissemination to all the organs. If it terminates it usually does so as a meningitis, either as a spilling into the ventricles of the brain from a belated overflow of a tuberculosis focus or a shower of bacilli that may occur at any moment from tubercles in the lungs or bronchial lymph nodes. In others this perifocal inflammation and tendency for hematogenous dissemination may be much less marked or last only for a brief interval.

The local lesion may range in size all the way from a few lobules of bronchopneumonia to many foci throughout both lungs, or to one large focus occupying most of one lobe or one

TABLE II. CHANGES IN VOLUME IN CUBIC MILLIMETERS IN THE COMPONENTS OF PRIMARY LESIONS AS AGE ADVANCED IN A SERIES OF KNOWN SEVERE CONTACTS

Age Period	Number of Cases	Average Volume Parenchymal Lesions	Average Volume Bronchial Node Lesions	Average Volume Tracheal-Bronchial and Tracheal Lesions
0-5	11	210	450	1350
6-10	9	87	555	805
11-15	9	31	92	137
16-20	10	33	73	12
20+	5	9	16	6

TABLE III. COMPARISON OF THE DURATION OF THE CLINICAL DISEASE AND LATENT PERIOD IN VARIOUS AGE GROUPS OF A SERIES OF SEVERE CONTACTS

Age Limits of Infection (years)	Number	Average Years of Clinical Disease	Average Years of Latent Period	Average Age at Death
1-5	14	6.1	18.5	27.1
6-10	18	3.4	12.6	23.5
11-15	23	2.8	7.8	23.1
16-20	14	2.1	5.9	25.5
20-26	9	4.0	3.5	30.0
Grand Average	78	3.5	10.0	25.8

lung. Dosage of bacilli has much to do with both size and character of the lesions.

If the patient survives, the lesions usually undergo a gradual retrogression with time. The perifocal infiltration thins out and disappears. The capsule gradually becomes heavier for eight or ten years and shrinks. After five or six years, resorption begins between the capsule and the calcified core and continues throughout life. At around the ten year period bone begins to form around the resorption areas, capillaries fill in, to be followed by lymphoid and marrow tissue formation. This usually becomes more marked in the majority of tubercles as age advances. Many of these local calcified lesions become entirely resorbed; others remain throughout life.

Adult Primary Lesions

When older people are infected for the first time, however, there are certain variations from these classical primary infections observed in children. Many of the lesions may be typical, as in children, but a large number are atypical in that they are usually found in the apex or along the subapical bronchi. They are usually rather small foci whose capsules are not well developed, and the calcification is not liable to be so dense. The lymph nodes towards the hilum are usually smaller and sometimes even lacking (Table II). As the capsules are usually not so well developed there is a tendency for these tubercles to spread by an overflowing process until they encroach upon a bronchiole or bronchus, after which there may be varying degrees of reinfection in the other parts of the lung which is ordinarily correctly viewed as a "reinfection" type of tuberculosis.

When these adult lesions fail to heal there is usually a more rapid evolution of the process from the primary to the reinfection type of

disease. While in infants and children this latent period may extend for ten to twenty years, it usually averages only a few years in adults. Perhaps the greater number of infections in adults do not develop into disease, but when they do there is usually more rapid progression than in children. It will be seen that this variable condition focuses active tuberculous disease in the young adult life. This is shown in Table III.

An important feature is that it is practically impossible to tell these adult primary lesions from the reinfection type *during life* because the lesions are usually small, out in the parenchyma, and poorly calcified. The tuberculin test is the only aid that is certain.

Primary Sequelar Lesions (Bernard)

The extent of progression to the "reinfection" type of disease from primary lesions is quite variable. In a recent study of close contacts, I found that nearly 75 per cent of patients coming to autopsy in our sanitarium, developed the disease directly or indirectly from the primary. In other communities, or in older age groups with more casual contacts, there is no doubt a much higher rate of exogenous reinfection.

The methods of spread from these first infections are also quite numerous and variable. In a large number of the lesions there is a rapid progress which is sometimes termed the *progressive primary tuberculous lesion.* The mechanism of this spread is usually not detected because it has destroyed much lung tissue before the pathological examination can be performed. Occasionally, however, the "breaks" in the defense mechanism can be found. Owing to the fact that there is no way of telling the ending of the primary and the beginning of reinfection, it seems logical to study them as one process, that is altered by allergic states, age, and to a less extent diet, race, et cetera. The local lesion may spread rapidly in pneumonic fashion or the tubercles may spread by "overflowing" the capsule.

As the disease progresses down the lymph channels toward the hilum there is a penetration of the perivascular spaces by toxins with a resulting tumefaction of all the adjacent lymph nodes. One reaction to this condition is a back-up or a stasis of lymph flow which on rare occasions may be seen on the x-ray picture as a gradual diminishing shadow from the hilum out to the base of the upper lobe. It may extend upward towards the middle of the lobe. This process may clear without leaving anything but a moderate amount of perivascular fibrosis (Case 1). On the other hand, it may cause a back-up into the tissues and result in a caseous pneumonia. The result depends largely upon the number of infectious microörganisms present. At the same time the process may extend through the bronchial wall into the lumen of the bronchus and set up a so-called bronchial catarrh. Bacilli may filter through the bronchial wall during this time and cause a positive sputum.

At this stage, a marked irritation of the bronchi frequently occurs, with a stimulation of the mucous flow that may be reflected clinically as varying degrees of severity of "asthmatic bronchitis." At first there is usually a marked increase of glairy mucus with mucopurulent flecks. Tubercle bacilli are not numerous, but present. Later inspissation of the mucus occurs and mucus plugs may form that occasionally lead to massive collapse of the lobe. This is sometimes not detected until it is seen on the x-ray picture. Atelectasis resulting from this form of collapse may be of varying degrees of severity. It may clear up rather promptly following the removal of the obseruction. In that event all that remains may be only a mild emphysema. It may also assist in the production of an effusion in the pleura.

Case 1.—A. I. was a thirty-two-year-old man of Russian descent. His mother was suspected of having had tuberculosis, and he worked as an orderly in a large general hospital just before his onset.

At the onset he had a pleuropneumonia for two days, following which he had fatigue and loss of weight. A few months later he had an acute attack with cough and a temperature of 103°. A diagnosis of tuberculosis was made on physical examination and x-ray. The sputum was at first negative but later was positive. There was a gradual recovery but occasionally a rise in temperature would occur.

The first x-ray revealed a slight left apical lesion with a small cavity also at the apex of the left lower lobe. The two points of origin of infection perhaps caused such a lymph node enlargement that there resulted a "back-up" into the upper lobe, as shown in the second x-ray. This cleared within the next few months, as shown in Figure 3.

In other instances there may be an obliterative caseous endobronchitis that leads to permanent

atelectasis. This is the condition that has been called by Bernard "lobitis," where there is a gradual contraction of the lobe towards the spine. The atelectatic lung undergoes fibrous contraction and adhesion to the mediastinal surface followed by a pulling up of the non-rigid lung below. This happens more frequently on the right side than on the left. The more severe degrees of involvement may disseminate a sufficient number of bacilli in tissues to cause a caseous pneumonia or a fibrocaseous tuberculosis. The mechanism of this will be described and illustrated.

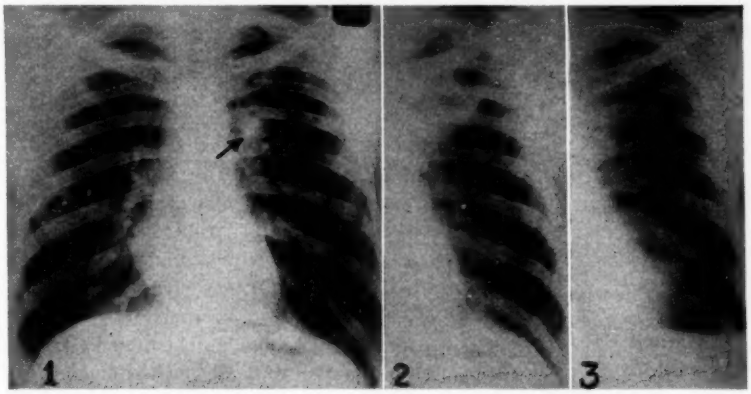

Fig. 1. Case 1. Roentgenogram taken January 12, 1931. Note beginning shadow in left hilum.
Fig. 2. Case 1. July 12, 1931—The infiltration has spread out towards the pleura.
Fig. 3. Case 1. January 13, 1933—Almost entire resorption.

The lymph nodes lying near a bronchus may compress the walls so that it is difficult for air to pass. Dyspnea with ronchi and sibilant noises are usually the result. If the inflammation extends into the bronchial walls there is a whole series of changes that are not ordinarily recognized. Belonging to this is the condition referred to by Ranke as "hilus catarrh." The toxins enter the wall of the bronchus and interfere with the secretion of mucus. It gradually becomes inspissated and may plug the bronchus, which is already narrowed by compression, in which case a temporary atelectasis results. If it does not clear within a short time, a permanent atelectasis may ensue with a resulting fibroid lobe.

Atelectasis of one lobe may consist of the so-called lobitis or sometimes the lobe may become infected with bacilli with a resulting caseous and fibrocaseous tuberculosis. All degrees of change exist between the two extremes. One case will be cited.

Case 2.—B. G. was a ninteen-year-old American woman at the time of death. There is nothing unusual in the history. The fact is there is no record of any illness except a pneumonia in infancy. She began to cough in November, 1936, and went to a doctor who treated her for "bronchitis." Another doctor told her that an x-ray "would settle the matter, but was unnecessary." Another could find nothing and dismissed her with the comment that she "was too well." In September, 1937, still another doctor annually became active and performed a tonsillectomy; but the cough, for which it was performed, did not improve, in fact it became productive. He then took an x-ray and diagnosed pulmonary tuberculosis.

Outside of the wholesome lesson to the medical profession, this case afforded a fine study of the effects of lymph node involvement on the subsequent course of the disease, including the clinical, x-ray and pathologic findings. A roentgenogram, taken on admission, showed a dense shadow along the spine about 2 cm. broad, from the apex to the hilum. There were infiltrative areas in the upper parts of both lungs, characteristic of tuberculosis. A roentgenogram taken a few months before death revealed a pneumothorax with a partial collapse on the right.

At the postmortem there was found an atelectatic left upper lung lobe and recent solid masses throughout the left lower lobe and the right upper. There were scattered acinous nodose lesions in the lower parts of the right. On sagittal section there was found a dry spongy left upper lobe and tuberculous masses, cavities, and nodules elsewhere. Forming a ring around the main upper lobe bronchus were three calcified tubercles of 5 to 7 mm. dimensions. The lumen was definitely narrowed.

There was a mild degree of bronchiectasis in the principal bronchi of this lobe. There were several scattered calcified foci throughout the lobe with the largest one about 5 mm., in the apex. There was a dry

spongy atelectasis throughout the parenchyma with no caseation and consolidation, and only a slight amount of fibrosis.

The microscopic sections revealed a uniform age character of all the calcifications consisting of a small central calcified core and an early specific capsule of moderate thickness. The middle capsule was rather thin. In an adult, the oldest tubercles corresponded to a four or five year process.

The parenchyma of the collapsed lobe was a rather uniform atelectasis with an occasional nodular tubercle, some of which had calcified centers.

The bronchi at the level of the lymph nodes consisted of a fibrosis extending in from the nodular tubercles towards the lumen. The mucosa was ulcerated in many places.

Comment.—This case illustrates a peribronchial lymph node phenomenon that led to atelectasis. Had this case been diagnosed in time, it may have been only a simple "lobitis," with uneventful recovery. The reference to the lapses in diagnosis are not given with any malicious intent, but to point out, in an earnest manner, one of the great needs in our medical education.

Finally, the lesions in the lymph nodes may become caseous and extend into the lumen of the bronchi, first as elevated nodular tubercles, then as open ulcers which may expel the lymph node contents with varying degrees of subsequent involvement. Much depends upon the richness of the growth of tubercle bacilli in the discharging node. There may be little or no subsequent disease, or there may be a diffuse terminal infiltration or acinous nodose spread throughout the lungs. Most of these are common and need no special elucidation. One mild result may be profitably shown.

Case 3.—R. C., a case of massive atelectasis, was a married woman, aged twenty-seven, who was in contact with a sister-in-law three years before entry into the sanitarium. The onset of the disease was insidious during the summer of 1938. She began to feel tired and sleepy most of the time, lost weight, and complained of a tightness in the chest, and that the wheezing was so marked it kept her awake. The sounds were in the expiratory cycle and could be "felt" along the left side of the sternum. A doctor ordered an x-ray examination (Fig. 4), which revealed tuberculosis, and her sputum was found positive for tubercle bacilli. She began to run a slight temperature and then suddenly experienced a sudden pain in the left chest, not very severe, but she promptly became short of breath. Her doctor diagnosed atelectasis.

On bronchoscopic examination by Dr. Sorboroff, there were observed several pearly nodules pushing up the wall of the left main bronchus, measuring 3 to 4 mm. across and about half closing the lumen. One was excised and was found to contain many acid-fast bacilli. The atelectasis gradually cleared.

Comment.—This is clearly a perivascular and peribronchial extension from the lymphatics, resulting in first a bronchial catarrh and then a tuberculous peribronchitis, with closure of the lumen either by pressure, actual growth, inspissated mucus, or all three. The node finally protruded into the bronchus causing a plugging with the subsequent atelectasis.

A similar growth may occasionally protrude into the pulmonary veins and produce a generalized miliary tuberculosis. It may also rupture into the mediastinum, into the pleural cavity, or through the esophagus. Extensions in the bronchi, however, are much more common.

When a late hematogenous dissemination results from any of these modes of spread, there are two principal types that are not only distinct in the mode of origin, but in the clinical end-results. They first become disseminated throughout the lungs and may not extend beyond them. Most of these originate from a lymphatic type of spread that involves the lesser circulation, through the thoracic duct and left subclavian vein. The other type involves the pulmonary veins which disseminate bacilli to the whole body. The former may be symptomless even though the x-ray may reveal showers of tubercles throughout the lung. The latter is usually more severe and commonly assumes the "typhoid" syndrome. Tubercles of this generalized type are usually most numerous in the lungs, second in numbers in the spleen, then they are found in order in the lymph nodes, liver, adrenals, kidneys, bone, meninges and rarely in the muscles. When they involve the meninges of the brain, there is the usual terminal meningitis.

In less severe cases of generalization, isolated foci may appear and ultimately become manifest as the various extrapulmonary forms of disease.

There is nothing constant, however, in bacterial hematogenous dissemination. Apart from the more constant "hematogenous phase" of the primary, bacteremia in tuberculosis usually appears in showers of different degrees of severity, depending upon the penetration of a capillary or larger blood vessel or the progression through the lymphatics long after the hematogenous phase of the primary.

Instead of an immediate progression, primary

tuberculous lesions may undergo gradual retrogression either with or without depositing daughter colonies locally or in the lymphatics. They may become quiescent for months, or even years. mining when an earlier infection exists. It is also difficult to determine the increase in the threshold of infectability caused by the existing infection.

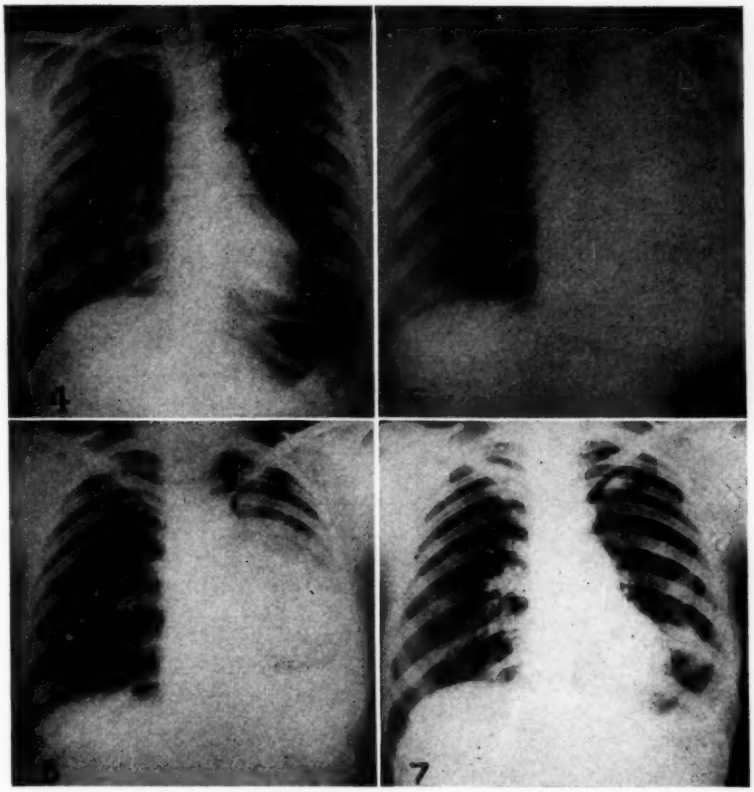

Fig. 4. Case 3. Roentgenogram taken November 8, 1938. Note density in left hilum.
Fig. 5. Case 3. January 3, 1939—Atelectasis of left lung. (Note the heart is pulled out of sight.)
Fig. 6. Case 3. January 17, 1939—Clearing.
Fig. 7. Case 3. April 18, 1939—Practically all cleared within about four months' time from onset.

Finally some of them or their slowly growing daughter tubercles may produce a result similar to those described for more rapidly progressive lesions. The slow progress of some tuberculous lesions is not yet sufficiently appreciated.

Exogenous Infections

Under the heading of exogenous infections, there are two distinct modes that should be recognized. The first is the possible infection that may occur on top of another older smouldering process. This has been properly termed a superinfection. It is difficult to detect such infections with certainty, because of the difficulty in deter-

The other type is a true reinfection, occurring on a completely healed process. Although it is practically impossible to distinguish between these two types, they will be considered together in the discussion on the development of the disease from them.

The parenchymal disease in these types usually begins in the posterior and upper aspects of the lung, most commonly along the subapical bronchi, less commonly in the apex, the horizontal rami, or in the apex of the lower lobes. When the process begins in the apices there is a tendency for the disease to be more benign in character.

Minimal Lesions

The size of the lesions at first may be microscopic, but, irrespective of the size at the beginning, the upper limit of size of a minimal disease has been defined by the tuberculosis associations. The first x-ray appearance may be in the form of tiny flecks barely visible. Undoubtedly the disease is present for some time before it shows even on the x-ray. They are observed more commonly in the first and second interspaces, with a slightly less common occurrence in the apex. If the Mantoux is first negative and turns positive, it is probably a primary infection in an adult. Without the Mantoux this distinction cannot be made, and with a positive Mantoux it may be the late phase of a primary. There are many more than ordinarily suspected. Several of these have been shown in a recent work where autopsies were performed.

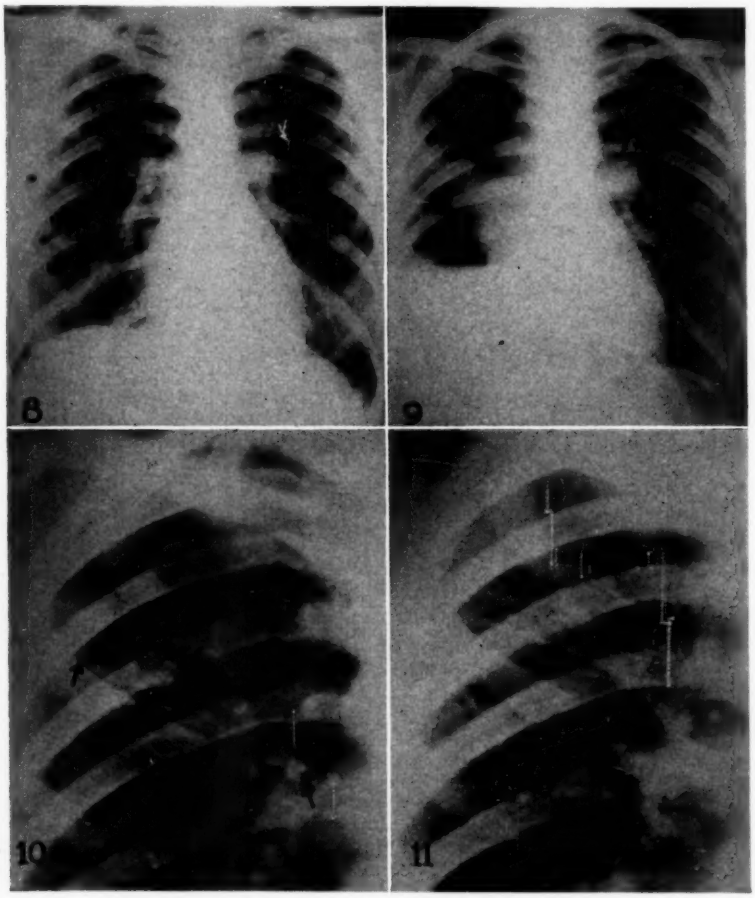

Fig. 8. Case 4. Roentgenogram of normal chest taken April 16, 1936.
Fig. 9. Case 4. April 1, 1939—Advanced disease, pneumothorax and effusion.
Figs. 10 and 11. Roentgenograms taken between Figures 8 and 9 reveal the development of the disease in the same case.
Fig. 10. Case 4. Sept. 2, 1937—Note minimal lesion in second right interspace, and calcified lesions at hilum.
Fig. 11. Case 4. February 23, 1938—Moderately advanced lesion in right upper.

Case 4 illustrates a small beginning that developed into a grave advanced lymph node type.

Case 4.—P. J. S. was a twenty-seven-year-old nurse who had a difficult service in a large tuberculosis sanitarium and at one time cared for an ill mother at night in addition. The x-ray studies reveal the changes that took place in her chest. At first there were a few tiny flecks in the right first intercostal space, then an increase in their size and number, and finally a positive sputum. Rest and a gain of twenty pounds in weight

did little good because the disease continued to progress. A pneumothorax was advised and given. The third picture showed a tiny cavity appearing and a moderately advanced lesion. She seemed to improve and was allowed to marry. The pneumothorax was continued and she seemed to be doing well. After a trip to Florida, however, she returned with a pleurisy and effusion and temperature from 99 to 103. The last x-ray (Fig. 9) is shown revealing the advanced conditions at present. The sputum and pleural fluid are positive for tubercle bacilli. She expectorated about 75 c.c. of glairy mucopurulent sputum at first, but this later became more purulent.

Comment.—This seems to be a progressive process, perhaps starting as a primary, as a small lesion in the parenchyma, and progressing to an ulcerative lesion with what appears to be a severe involvement of the lymph nodes around the hilum causing effusion, cough, mucoid sputum and dyspnea. Later the sputum changed to a more purulent type, apparently after the bronchi became more caseous and the mucous glands became impaired by the toxicity. This case passed through the moderately advanced to the far advanced stage.

As the lesion becomes older the flecks may become encapsulated. At no time does the minimal lesion itself produce any grave systemic symptoms.

In the late stage these flecks may spread to smaller infiltrates, but usually they become encapsulated and heal, leaving behind some form of calcified focus. Some of these forms may have been included by Puhl, Simon and Stefko, in the foci bearing their names.

Moderately Advanced Lesions

In the moderately advanced disease the first signs are infiltrates of varying sizes, most of which are extensions from minimal lesions. They may have perifocal flecks and may extend over several rib interspaces on the x-ray picture. There may or may not be clinical symptoms.

Some of these infiltrates may go on to ulceration and produce a peribronchitis involving the hilum lymph nodes, and there may be a condition similar to what has been described under the previous headings of a sequel to a primary infection. In the late phases of this stage of disease there may be an extension to fibrocaseous and ulcerative disease that passes into far advanced type. Nodular, miliary, acinous-nodose tubercles, and round infiltrates may follow these various lesions. *As the disease progresses further, there is a tendency for the lesions to appear in bronchi in the anterior part of the lung.*

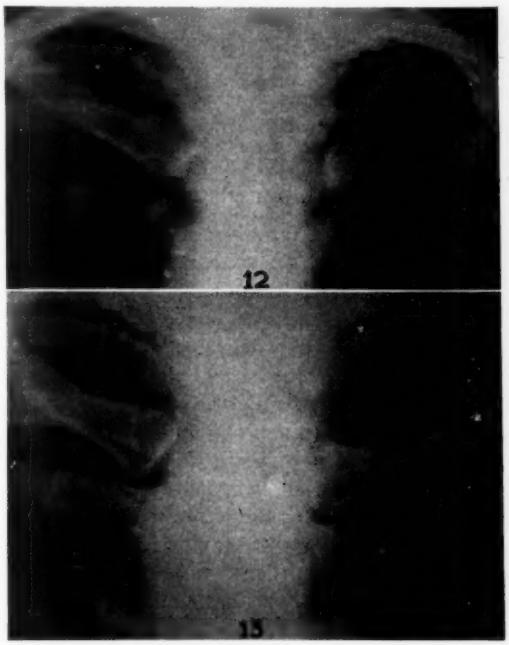

Fig. 12. Case 5. Roentgenogram taken June 9, 1926. Note small cavity in left apex with two small calcified lesions.
Fig. 13. Case 5. March 2, 1938—A quiescent fibroid lesion in both apices—more in left.

Without any signs to indicate it, these lesions may go on to healing, produce fibroid cavities, clearing acinous-nodose and nodular lesions, and leave behind an emphysematous residue in the parenchyma and dilatation of the bronchi as a bronchiectasis. One such case will be cited.

Case 5 illustrates a moderately advanced lesion healing spontaneously or at least advancing very slowly.

Case 5.—C. H. was an assistant housekeeper in a large tuberculosis hospital for twenty-four years. She was about forty-five years old, with no complaints at any time referable to the lungs. There was a routine x-ray taken in 1926 (Fig. 12) revealing a small cavity in the left apex with two calcified lesions in the wall and one or more small calcified lesion towards the hilum. There was also a slight spread to the right apex. The lesion was perhaps an adult primary six to ten years old. It must have been six years old to show the amount of calcium present, and it probably was acquired on duty and would not have been over twelve years old, more probably not over ten. The x-ray (Fig. 13) taken twelve years later (and about twenty

years after infection) revealed a slight spread of the disease, but it was definitely more fibroid and, barring accidents, was on its way to a spontaneous healing.

Comment.—This appears to be a small apical adult primary infection, spreading slowly to a fibrocaseous, fibrocalcareous and fibroid disease over a period of more than twenty years. At no time were there clinical symptoms, and it is likely that no clinical disease ever will result if the patient lives her usual life.

Far Advanced Lesions

Last, there is the far advanced stage which has the same phases as the others, except there is a greater involvement from the beginning. The infiltrates may be massive, involving the larger part of the lobe; may be pneumonic, involving one, two or more lobes; and there may be an extension to the lower lobes. It may also result from a slow progression of minimal and moderately advanced lesions, as in Case 4.

As the disease becomes more chronic many months to years after the onset, the lesions may become more fibrotic; cavities which have formed may develop dense walls and even disappear. There may be an equilibrium established between the host and the disease which lasts from years to decades. *Like the moderately advanced lesions, as the disease progresses, the lesions become progressively caudalward and anterior.* This is the type that has filled most of our sanitarium beds in the past.

The late period of this far-advanced stage may be a terminal process that is manifest by an extension to the bases and an involvement of practically all the good lung tissue. "Gravity" infiltrates may form along the ends of the lower lobe bronchus that resemble clusters of grapes. Lobal patches of caseous bronchopneumonia may appear in the lingula or in the other marginal aspects of the lung. Sometimes a caseous pneumonia will involve one of the lower lobes. Extensive fibrosis will be found around the older cavities with marked contraction.

On the other hand, even these advanced stages may heal, leaving behind fibroid scars in place of cavities or very heavy walls surrounding old cavities with fibroid peribronchitis and fibrous scars in place of what were formerly acinous-nodose and nodular tubercles. In every case, however, where healing takes place, as just described, there is a residue of emphysema that plays a very important rôle. It may be rather diffuse or it may be bullous in character. Irrespective of type it usually cuts down the capillary bed and leaves the patient dyspneic. Extreme cases of this type may go on to right heart failure.

Case 6 illustrates a clinically silent form of tuberculosis going on to a terminal emphysema.

Case 6.—F. H. C. was born in England and came to the United States at the age of eight. There was no tuberculous history. He was a fifty-nine-year-old man at the time of death in December, 1938.

He finished the seventh grade in school and worked as a clerk and railroad passenger agent for thirty-eight years. In 1931 he stopped work on account of ill health, which dated back to 1928.

His past history revealed that he had gonorrhea in 1920 with stricture, and syphilis in 1922, which was treated one year and pronounced cured. He had a fistulectomy in 1926 at the Mayo Clinic for a fistula "of twenty years' duration." His onset was with cough in 1929, but he had not been well for some time. The symptoms were, cough for some time, expectoration one ounce in twenty-four hours, slight blood streaking once or twice, loss of ten pounds in weight, and dyspnea for a year.

The physical examination as reported was as follows: "General development fair; general condition fair; skin clear; teeth two plates; fingers clubbed; heart within normal limits; heart sounds first loud systolic and presystolic; murmurs at apex, presystolic thrill; blood pressure 140/76."

Chest examination: "Impaired expansion bilaterally; dullness over upper third bilaterally; right, varied râles and harsh breath sounds; increased whispered voice in upper half; left, similar findings. Diagnosis—pulmonary tuberculosis, far advanced "B"; multiple cavitation bilaterally."

Electrocardiographic report: "Heart rate 110; rhythm irregular due to extrasystoles. QRS 1, 2, 3 slurred. QRS 1 low. ST 2, 3, depressed. Diagnosis—Myocardial damage; auricular premature contractions."

X-ray examination: "Apices are both very hazy. Diaphragms are oblique. Costophrenic angles are both obliterated. Cardiac shadow is enormously enlarged both to the right and left. There is an infiltration of both upper fields, characteristic of tuberculosis, and a definite enlargement of the heart."

The essential laboratory findings revealed 150 mgs. albumin per liter in the urine; a slight shift to the left in the blood picture, and negative Wassermann and Kahn tests in both blood and spinal fluid. All else was essentially negative.

The essential postmortem findings were cyanosis, mild edema of the ankles, a very large heart (weight 415 gms.), with a right ventricle measuring 9 mm. in thickness. The left ventricular wall measured 13 mm. The musculature was soft and pale yellow-brown.

The analysis of the postmortem x-ray of the lung was as follows: There were scattered calcifications in both upper lobes up to 6 mm. The bronchopulmonary nodes showed calcifications up to 4 mm. There was a

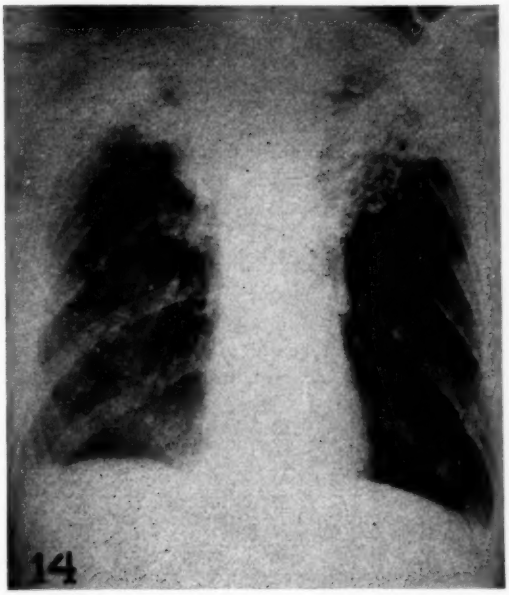

Fig. 14. Case 7. Roentgenogram showing fibroid uppers, emphysematous bases.

fibroid 3 cm. cavity in the left upper and scattered acinous-nodose and patchy pneumonic foci throughout both lungs. There were extremely large bullæ along the diaphragmatic and anterior margins reaching the size of 10 cm. The tracheal cartilages were calcified.

This case represents an emphysema of healing as well as a marked marginal emphysema due to effort.

Comment.—The date of origin of the tuberculosis is not known, but must have been before 1906, the approximate date of the beginning of his fistula, which presumably was tuberculous. The calcifications and fibroid lungs also point to a disease of twenty or more years duration.

The probability is that the infection was a young adult or childhood type that was essentially symptomless throughout life, but gradually progressed and resulted in a fibroid upper part of his lungs with a gradual developing emphysema in the lower parts. This caused an extra burden on the right heart and resulted in the myocardial hypertrophy and damage.

Case 7 was a disease advancing throughout life with dyspnea the first significant recognized symptom.

Case 7.—W. H. was a sixty-year-old American painter and decorator of Irish ancestry. The patient was nine years old when his mother died of pulmonary tuberculosis after two years of illness. He was perhaps infected when about five to seven years of age, or fifty-three to fifty-five years before death. He worked hard all his life, drank heavily, and raised eleven children. He had pneumonia (tuberculous?) at thirteen, twenty-six, and forty-two years of age. At twenty he had a pulmonary hemorrhage, diagnosed as "asthma." At fifty-five he had a severe pulmonary hemorrhage and began to raise sputum in which the health department found tubercle bacilli. He came into the sanitarium as a result of loss of weight (twenty pounds), weakness and dyspnea.

The clinical examination revealed "an emaciated old man, with a slightly barrel-shaped chest. He breathed with the accessory muscles of respiration. On percussion there was hyperresonance below and dullness in the upper part of the chest. Auscultation revealed absent breath sounds, prolonged expiration with wheezes and ronchi, moist râles in the anterior part of the chest, and whispered pectoriloquy in the apexes."

The x-ray revealed multiple fibroid cavities in both uppers with a retraction upwards and a marked emphysema in both bases. The heart was enlarged.

The sputum was positive, although many such cases become negative, years and even decades before death.

Extrapulmonary Tuberculosis

The extrapulmonary localizations may be in the form of miliary spread, as mentioned before, coming in the wake of the primary or as a later "accident." It may involve the pleura, mucous surface of the bronchi, trachea, larynx, and gastrointestinal tract by direct extension. The genito-urinary tract may be involved by foci borne by the blood stream, also the various other extrapulmonary foci including the spleen, kidney, liver, adrenal, bone, skin, meninges, et cetera.

Atypical Phenomena

There is included in this classification a special group of cases that are markedly controversial as to cause and clinical findings. Without wishing to complicate the literature, I have used the term *paratuberculosis*, although it has been used before in another sense. These are the forms of the disease that I believe may be explained on the basis of a filtrable or atypical form of the germ. These phenomena are perhaps associated with the many cases of classical tuberculosis, but in their pure forms they are relatively rare. I believe we may consider part of the cases of polyserositis, Pick's cirrhosis and erythematous lupus belonging here; also Poncet's rheumatism, Per-

the's disease and perhaps Schaumann-Besnier-Boeck's syndrome, and certain types of erythema nodosum, and disseminated follicular lupus.

Complications and Sequelæ

Another grouping includes those cases that may be considered as complications and sequelæ such as pulmonary hemorrhage, fatty and amyloid changes in the parenchymatous organs, emphysema, bronchiectasis, et cetera (Cases 6 and 7 illustrate emphysema).

Associated or Concomitant Diseases

Finally, we may consider diseases associated with tuberculosis but not related to it. Although they may play no rôle as a cause of the disease, yet they must be reckoned with when present. Such diseases are carcinoma of the lung, or, in fact, carcinoma elsewhere in the body, with secondary metastasis to the lung; diabetes, syphilis, heart disease, kidney disease, and practically every condition known to medical science.

Effect of Immunological Factors on Morphology

After visualizing these many anatomic changes in tuberculosis, some of the fundamental causative factors may be reflected upon in order better to understand the clinical results and to lay a basis for effective therapy and a better public health control. What brings about the various changes described? What brings about clinical symptoms? These factors are not all known, but I believe enough is known to divide them into specific and non-specific types, each of which may be discussed.

The non-specific elements, such as protein split products, toxic amines, etc., result from any tissue destruction and perhaps enter into most disease processes. Whatever they may be they injure the normal body cells, causing a deranged metabolism; lead to nausea, loss of appetite and then loss of weight; interfere with the heat regulating mechanism, causing a rise in body temperature; irritate the neurogenic heart centers, increasing the pulse rate; in fact, the whole normal mechanism is deranged so that an abnormal physiology is the result. These may be termed *general* or *systemic* effects, in contrast to local effects which result from any intercurrent episode such as effusion, hemorrhage, ulceration, etc.

There is demonstrable evidence of specific effect in the form of tuberculin reaction and humoral or cellular elements to neutralize it. There has also been shown a decrease in allergy or a desensitization due to a gradual increase in antigen.

The attempts to correlate tubercle information, cytology and pathological reactions with allergy, however, have heretofore met with failure or have become controversial, but there is undoubtedly a relationship. In the primary lesion there is a different reaction than in the late stages of the disease. But whether the "secondary" tuberculous process is a regular function of the allergy-immunity complex is still obscure. There is much to support it. The complex of allergy and immunity varies greatly with age, race and even in individuals. Some day it may be linked up with the changing cytology of the disease. In slow forming tubercles or in people with low allergy, or in a desensitized lung, the tubercles seem to resemble more the primary type of lesions. On the other hand the rapidly forming types resemble abscesses.

One of the interesting anomalies of tuberculosis and one that has in the past, and still does, offer grave obstacles to control, is the fact that there is no consistent relation between clinical symptoms and morbid anatomy. Extensive disease may be present without symptoms sufficiently severe to send the patient to a doctor or for the doctor to find it without any x-ray picture or careful laboratory studies. So light are symptoms in some cases that a considerable percentage become spontaneously healed and are never found unless an autopsy is performed.

Whatever may cause this disparity cannot be given for the very good reason that it is not known, but there must be some relation, however, to the absorption of toxins.

It seems logical that there is either a walling-off of the toxins or a neutralization of them, or both acting together in symptomless disease. The toxic substances do not seem to reach the vital centers in quantities sufficient to cause a derangement of normal physiology. In such cases, the cavities expel mostly all their toxins with the sputum. Only a slight amount of absorption takes place from the heavy granulation tissue

around the cavity well. There may be just enough to increase the destruction of old polymorphs and to increase the young cells, to change the colloid stability of the blood and increase the rate of fall of red blood cells; but not enough to raise the body temperature, increase the pulse rate or lead to a perceptible loss of weight. As a consequence there may be only a slight leukocytosis or a slight "shift to the left," or a slight increase in red cell sedimentation, as the only indication of disease.

An infiltrate or a cluster of tubercles may be so well encapsulated that the same result occurs, although there may be a slow "leaking" of bacilli through the capsule that, over the years, may spread and ultimately result in active disease.

Importance of Dosage

What is of supreme importance, however, in the disease as a whole, seems to be the *size of the dosage of bacilli at the beginning of the infection, and the subsequent multiplication of them in the bodies of the infected patients.*

All rules seem to be suspended when huge doses occur for the first time. Violent acute inflammation results rapidly. The sheer quantity of material overbalances all natural defense processes. Excesses of nature's own cells, living and dying, aggravate the process. At first it is nonspecific and independent of allergy, but as allergy appears later the untoward effect is multiplied. Large dosage either with or without allergy seems, therefore, to be the main cause of the "abscess" type of tubercle and rapid progression.

While a mild allergy itself may assist in protection, it seems to be on the whole a trouble maker so far as spreading the disease is concerned. Above all, it should not be looked upon as a synonym for immunity, for the two are in certain respects separate mechanisms. This is apparent when a loss of allergy may occur either artificially or during the course of the disease, yet resistance is maintained. It does seem to be largely responsible for the changing character of the tubercle from the epithelioid-fibroid type to the polymorph-lymphocyte-abscess type. The resulting disease would appear to be much of a function of this interesting relationship.

Variations of Parasites and Its Relation to Pathology

A word may also be said about the tubercle bacillus and its variants and the bearing they may have on human disease. This offers a new "frontier" in the tuberculosis problem. There are variations of the tubercle bacillus that produce unusual pathological effects. There seems to be good evidence that very small or even filtrable forms may play a rôle in the disease.

The course and change of the germ in the body is still only vaguely known. Acid-fast bacilli rarely grow abundantly in the tissues. On the other hand, in freshly opened up cavities, they will not infrequently grow in masses, and in such cases patients are not only a menace to themselves, but to everybody around them.

The Relation to Public Health

This brings in the public health aspect as related to the pathology and bacteriology. Like clinical findings, there is little relationship of the extent of pathology to the numbers of bacilli. They depend usually upon an ulcer or cavity.

The most important factor for the public is, therefore, the presence of *sputum that is positive for tubercle bacilli.* The next in importance is the *number of bacilli present per cubic centimeter of sputum.* They usually vary in proportion to the amount of surface of recently excavated caseous lesions. The fresh caseum seems to act as a culture medium in which bacilli sometimes grow in masses. The third important feature is the ability of the bacilli to grow in the tissues. This may be quite properly termed *virulence.* The causes of the growing of bacilli in tissues, with their non-specific and specific effects, however, are still unexplored frontiers in tuberculosis.

Not all positive cases, however, are of significance so far as the public health is concerned. Tumescent lymph nodes may allow bacilli to "leak" through into bronchi for three or four years after a primary lesion has been healing, and bronchial ulcers may expel bacilli. In such cases there is little sputum, and the germs may only be detected on stomach lavage or bronchial swabbing. The bacilli in such a case are, therefore, important to the individual but not to the public health.

Before closing, something should be said about the relationship of these many phenomena to

therapy. The object of a rational therapy in addition to the time-honored food and rest, should be first a system of prophylaxis by removal of the open and occult sources of bacilli. In vulnerable young people it may be expedient to protect by vaccination. In known cases of tuberculosis, treatment should aim to decrease the growth of bacilli in the body by a neutralization of the toxins, both specific and non-specific, and to increase encapsulation. Finally, it should aim to cut down the amount of sputum. These objectives are best accomplished by rest, a balanced diet and wherever possible the use of collapse therapy measures.

In conclusion, I wish to emphasize that the rather terse declarations in this thesis may appear as unwarranted dogmatism, but it is a well attested fact that practically all of the assertions made regarding essential principles, represent classical studies of a vast number of workers. Reference to all these would be prohibitive and really inexpedient in this type of presentation, intended as it is for ready reference of medical men in general practice. It should be pointed out, however, that a better understanding of the new aspects of the problems has been greatly facilitated by a meticulous study of over 1,200 autopsies on our service at the Chicago Municipal Tuberculosis Sanitarium. The classification presented is not to be construed as anything to be used at once, or, for that matter, ever to be used. It is only intended as a temporary framework around which the facts concerning this complex disease may be "draped," the better to understand the disease myself and perchance to help convey that understanding to others.

THE TREATMENT OF VAGINAL DISCHARGES*

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ABNORMAL vaginal discharge of a non-sanguinous nature is one of the most common gynecologic complaints and yet it is probably treated more inadequately generally than any other similar symptom. Recent advances in the study of leukorrhea have elucidated many etiologic problems and have made possible more intelligent and more effective treatment. The application of this new information to the actual therapy of the condition demands the utilization of certain simple bacteriologic procedures and has made ordinary symptomatic treatment archaic. There are admittedly many gaps in our knowledge, but this does not condone failure to use the data that are available. In the time available, I shall attempt to emphasize those facts of physiology and pathology which are best established and most useful.

Under completely normal conditions the vaginal discharge is sufficient only to keep the walls of the canal moist. The fluid portion of the discharge presumably represents the minimal secretion from the cervical glands, to which are added masses of epithelial cells desquamated from the mucosa and aggregations of the vaginal bacilli. The mucous membrane itself possesses no glandular elements and consequently is generally said to have no secretion, although it is probable that a small amount of fluid may diffuse through the mucosa and provide the necessary moisture. In any event, individuals who have been subjected to total hysterectomy rarely complain of unusual vaginal dryness even though there can be no cervical secretion. The vaginal bacilli of Dœderlein react upon the glucose and glycogen provided by the epithelial cells to produce lactic acid, which maintains a considerable acidity in the discharge and largely prevents the growth and development of pathogenic organisms.

There is considerable excellent evidence that the vaginal mucosa and the cervical glands are under the control of the female sex hormone. In the absence of this estrogenic stimulation, the mucous membrane becomes thin and easily traumatized, the vaginal bacilli become less plentiful or disappear and the acidity is reduced—conditions which favor the development of an abnormal flora and predispose to infection with pathogenic organisms. The cervical glands likewise are stimulated to growth and activity by estrone, and regress when deprived of its effect.

The amount of the vaginal discharge may oc-

*Read before the annual meeting of the Minnesota State Medical Association, Minneapolis, June 1, 1939.

casionally be increased by physiologic stimuli and may cause some concern to the woman. The most common instance of such an increase occurs as a result of sexual excitement, when there appears to be a greater output by the cervical glands and the vulvo-vaginal glands of Bartholin contribute their mucous secretion at the introitus. It is also well recognized that many healthy women are conscious of a premenstrual increase in the discharge which is probably best explained by assuming a hormonal stimulation of the cervical glands, together with pelvic hyperemia, since it is unassociated with any apparent abnormality. Finally, the leukorrhea of early pregnancy, which may appear so abruptly and be so profuse as to suggest an acute gonorrhea, is also best explained on an hormonal basis. In such instances, the absence of pathologic findings should suggest the proper diagnosis and should serve as a basis for reassuring the patient, but only after thorough examination for other factors has been negative. It is not wise to assume that a leukorrhea is physiologic merely from the fact that its appearance coincides with the period when such discharges are known to occur.

The clinical study of any patient with leukorrhea should follow a well organized plan and it should be remembered that the discovery of a single etiologic factor does not necessarily remove the possibility that other pathogenic agents may be present. A careful history will elicit information about the time relationship to the menses, which may be important, and will determine whether the discharge is irritating during any portion of the cycle. In suspected gonorrhea, a history of possible exposure is obviously significant, and in the case of children the questioning should include the various details of family life which might give a clue as to the source of the infection. In adults, a pelvic bimanual examination should be carried out to detect gross genital disease, and the cervix should be inspected carefully through a speculum with satisfactory exposure and adequate illumination. Finally, bacteriologic examinations with smears or spreads, hanging drop preparations, and cultures should be done, in an effort to determine the most probable etiologic factor. It is generally recognized that leukorrhea usually originates in the lower genital tract, including the cervix, and that uterine and tubal discharges are relatively very uncommon except in malignant disease. The older concept of chronic endometritis is untenable in view of our modern knowledge.

Chronic cervicitis, undoubtedly the most common cause of leukorrhea, presents various manifestations, which to some extent dictate the form of treatment. Under this classification, it is usual to include the erosions and the eversions, and possibly the cervical polyps.

Cervical erosion is generally looked upon as an evidence of infection, although there are some investigators who believe that there is an underlying hormonal factor, since it may appear in virginal women or in young girls with no stigmata of bacterial invasion. On inspection, the cervical portio presents a greater or lesser absence of the usual covering stratified squamous epithelium. The cervix appears fiery red and the red area, which is sharply defined from the vaginal mucosa, does not stain with iodine. In non-infected individuals, the surface is smooth or slightly granular, while in those with true endocervicitis enlarged nabothian follicles may protrude from the surface. The eroded area, which is covered with cylindrical epithelium or has no epithelial covering, usually bleeds slightly on contact and there may be a history of spotting following douching or coitus. When the erosion is of the so-called "congenital" type, it is doubtful whether any of the usual methods of therapy are effective. On the other hand, erosions resulting from infection can be treated by applications of strong silver nitrate solution (25 per cent) or by brushing the surface gently with a cautery and by puncturing the visible follicles. In some instances, it is necessary also to cauterize the cervical canal, but ordinarily surface treatments will suffice. Healing is accomplished by stimulating the growth of the stratified mucosa up to the external os, a development which necessarily takes time, and may require a series of light cauterizations at intervals of a month or more. Patience will usually avoid the necessity for deep burning or for surgical intervention. As healing occurs, the discharge usually lessens, and it is difficult to persuade the patients to continue treatment until the lesion is completely cured.

Cervical eversions occur in women who have had cervical tears, either from an obstetric episode or from surgical dilation of the cervix. The cylindrical epithelium of the canal thus becomes exposed and is more susceptible to bac-

terial invasion. In the milder cases, silver nitrate applications or surface cauterization will promote the growth of squamous epithelium down to the new os, but when the lesion is more extensive it may be necessary to do a cervical amputation, a trachelorrhaphy, or a conization. Generally, the cautery will provide some relief, and there are many who believe that the surgical attack should be postponed until the conclusion of childbearing.

Cervical polyps produce leukorrhea, which is commonly associated with "spotting," but in some cases there is no bleeding. The lesion is easily diagnosed by inspection, when the bright red polyp is seen protruding from the os. Such polyps are generally benign and may be removed by severing the pedicle with the cautery or scissors, or by twisting it off. When patency of the os suggests that there may be other similar growths higher up in the canal, and especially in older women, it may be advisable to perform curettage and to have the material subjected to histologic examination.

As a rule, the leukorrhea associated with cervical lesions does not produce irritation unless there is a complicating vaginitis, due to the invasion of certain specific organisms of which three—the gonococcus, trichomonad, and monilia —are of special importance. Occasionally, the type of discharge and the history suggest the causative agent but in the final analysis the diagnosis must be made by microscopic or bacteriologic examination. Gonorrhea is suggested by the sudden onset with a profuse purulent discharge, by urinary urgency and frequency, and by apparent involvement of Skene's or Bartholin's glands. In trichomonas infections, the discharge tends to be yellow, limpid, and frothy, and symptoms are aggravated in the post-menstrual period. Monilia are more apt to produce premenstrual irritation, the discharge tends to be thick and white, and thrush-like patches may be visible on the mucosa. In general, however, it is dangerous to rely on such observations, particularly because mixed infections are relatively common and because any type of vaginitis may closely simulate the others.

Gonorrhea may be diagnosed by stained smears or, preferably, spreads, which reduce the risk that the cocci will be extruded from the leukocytes. Simple methylene blue staining may suggest the proper diagnosis, but only a well made Gram preparation will be conclusive. The biscuit-shaped organisms must be intra-cellular if the diagnosis of gonorrhea is to be made. Within the past few years, cultural technics have been developed which are much more sensitive than stained preparations. In doubtful cases, this diagnostic procedure should be employed even though it entails sending the patient to a properly equipped laboratory. The gonococcus is so sensitive that cultures must be incubated almost immediately at the proper temperature.

In adults, gonorrheal vaginitis is only a transient and insignificant part of the clinical picture, and consequently need not be treated for itself. Lower tract gonorrhea as a composite entity is, however, very important because of its tendency to involve the internal genital organs by direct extension, and demands the best and most logical therapy available. The older practice of employing douches and topical applications in the hope of killing off the gonococci by direct exposure has been largely abandoned, because it apparently does more harm than good. Reliance is now placed on rest, local cleanliness, and the administration of members of the sulfanilamide group of drugs. It appears that the results are not so good as in the male, but that they are better than previously.

In contrast to the situation in adults, gonorrhea in prepubertal girls and in postmenopausal women frequently produces a true vaginitis and rarely invades the upper genitalia or the vulvovaginal glands. It is generally agreed that the susceptibility of the vaginal mucosa in these age groups is associated with changes in this membrane incident to the absence of stimulation by the estrogenic hormone. At birth the mucosa, which has had the intrauterine benefit of transplacental estrone, is adult in character and so immune to gonococcal infection that gonococci introduced during birth are unable to gain a foothold. Within a few weeks this immunity disappears with the thinning of the mucosa and the decrease in the acidity of the discharge. Practically the same situation develops after the menopause, and evidently for the same reason. In both age groups, the mucosa becomes easily infected by various organisms, including particularly the gonococcus. Irrespective of the infecting agent, modern therapy includes the administration of the estrogenic hormone in an attempt to increase local tissue resistance. In

young girls, the gonococcus is the chief offender, while in postmenopausal women other organisms are more commonly involved.

In either instance, treatment consists in simple local cleanliness, and the administration of the hormone either intramuscularly in oil or preferably in the form of vaginal suppositories. Under this stimulus, the mucosa assumes an adult character and eventually becomes so resistant that the organisms die from lack of proper environment. Care must be enjoined in carrying out such treatment, so that other structures under control of the hormone, particularly the breasts and uterus, are not stimulated too much. The dosage of the hormone will, of course, vary with the size and age of the patient, but very large doses are to be avoided. Uterine bleeding and undue enlargement of the breasts indicate reduction in the dosage or the temporary discontinuance of treatment. It has also been argued that the administration of rather large doses of Vitamin A is both logical and useful, especially in senile vaginitis of any variety, since this accessory food factor plays a rôle in epithelial growth. In gonococcal infections, it is also advisable to give sulfanilamide, although its effectiveness in this condition has not been conclusively shown.

The *trichomonas vaginalis* is known to be associated with irritative vaginal infections, but its etiologic significance is not yet clear. There are some investigators who still believe that it produces the vaginitis, whereas the majority agree that it is probably a secondary invader without etiologic significance. Attempts to solve this problem have so far been ineffectual because of the extreme difficulty in obtaining the organism in pure culture, a feat which has not yet been accomplished in spite of persistent efforts. In the meantime, other organisms, especially the streptococcus subacidus, have been assigned the etiologic rôle, but these findings have been disputed to the point where the whole question is still quite unsettled. In any event, women with this infestation, who present irritative symptoms, must be treated in spite of the existing scientific uncertainty. Many individuals harbor the organisms without symptoms, a fact which further complicates the situation.

The diagnosis of vaginal trichomoniasis can be made by examination of the fresh discharge or by culture. For the former, some of the material adherent to the vaginal wall is suspended in normal salt solution on a cover-slip and placed on a hollow-ground slide. The trichomonads, which are somewhat larger than leukocytes but smaller than epithelial cells, are detected by their motility, which is apparently preserved throughout their life. The cultural technics that are available are rather difficult and uncertain, but in the hands of those with experience frequently detect organisms missed on examination of the fresh material. It is, however, doubtful whether such mild infestations are usually associated with symptoms. It should be kept in mind that the trichomonads do not stain easily and cannot be found in ordinary smears or spreads.

Apparently, trichomonads are never present in discharges which show a normal vaginal flora—other organisms than the Dœderlein bacilli are invariably present. Because of this fact some observers believe that the associated bacteria actually produce the symptoms, and consequently direct their treatment at alterations in the flora, in the hope that the reëstablishment of normal conditions will make the medium so unfavorable that the parasites will perish. The trichomonads cannot survive at the normal vaginal pH, but find conditions increasingly favorable as neutrality is approached—a circumstance which also affects the contaminating bacteria in similar fashion. The vaginal acidity may be increased by frequent douches with a mild acid solution—lactic acid or acetic acid in the form of vinegar. In addition, a sugar, dextrose or lactose, can be introduced to encourage the growth of the vaginal bacilli which remain. Attempts at actual inoculation of pure cultures of the vaginal bacilli have not been very successful, even though the maneuver has much to recommend it.

The alternate and more popular methods of treatment aim at destroying the trichomonads and the organisms living in symbiosis with them. Various therapeutic agents have enjoyed temporary reputations but none has proved uniformly successful, since there is nothing specific in their application. At present insufflations of silver picrate or of devegan powder are in vogue, and appear to be better than the simpler antiseptics and dyes formerly employed.

Finally, Hibbard and Falls have introduced a form of serologic therapy based upon their belief that the streptococcus subacidus is responsi-

ble for the symptoms of the infestation. The general immunity to this organism is raised by subcutaneous injections of increasing amounts of a vaccine and supplemented by local applications of a filtrate of streptococcus subacidus cultures in broth to the vagina. The originators of the method claim excellent therapeutic results, which independent observers have been unable to duplicate. Moreover, there is still considerable disagreement concerning the identity and the etiologic significance of the streptococcus subacidus.

It can, perhaps, be said that this method of treatment is still *sub judice,* but that further work along this line may develop a truly useful and valuable procedure.

The source of the original infection and the factors which occasionally make it so resistant to treatment are yet unsolved. It is well known that trichomonads are frequent and harmless invaders of the mouth and lower gastro-intestinal tract, and, in men, of the genitourinary organs, especially the prostate and seminal vesicles. Considerable investigation has been undertaken to correlate these facts with observed vaginal infestations, but the relationship is still not clear. It has been emphasized that the vaginal organisms are slightly different morphologically from those observed in other foci, and it has been suggested that these structural changes may be induced by alterations in environment. In any event, the possibility remains that certain patients may appear refractory to therapy, when the continuance of the disease is actually occasioned by repeated reinfections from their own hollow viscera or from sexual contacts. Such possibilities should be borne in mind whenever therapeutic refractoriness is observed, and should direct attention to certain obvious details of personal hygiene.

The *monilia,* a simple yeast form, has more recently been accepted as a definite etiologic factor in certain cases of vaginitis, which may occur at practically any age, but is more common during sexual maturity and especially during pregnancy. It is also undoubtedly the cause of diabetic pruritus vulvæ, although this revised concept has not yet completely replaced the older orthodox belief that the sugar-laden urine is irritating in itself. The monilia thrive best in an acid medium and consequently are most likely to produce symptoms in the pre-menstrual period. They may, like the other so-called specific organisms, be harmless invaders, or they may produce the most severe vaginal irritation that is observed clinically. Those who harbor the yeasts may experience repeated attacks of vaginitis over long intervals.

The monilia can be easily obtained in pure culture, and human inoculation experiments have demonstrated that they actually produce the irritation with which they are associated. It is, however, not yet clear why some women may harbor them for years without symptoms. Attempts to explain these vagaries on an allergic basis have not been eminently successful, although there is some evidence that certain individuals may have a developed sensitivity. Allergic skin tests have not generally correlated with clinical findings.

Yeast vaginitis may be suspected when the discharge is flecked with small white particles or when white flakes of the growing fungus are detected on the vaginal mucosa in thrush-like patches. The actual diagnosis must, however, be made from stained smears or from cultures. The monilia stain easily with the usual bacteriologic dyes and are very Gram-positive. In such preparations they appear as dark homogeneous oval forms, the conidia, or as granular threads, the mycelia, which may be seen to branch. Occasionally, these structures may be detected in fresh unstained material, when the conidia appear as small highly refractile particles and mycelia as definite threads. Nevertheless, it is always advisable to resort to culture for the final diagnosis. The monilia grow abundantly on slants of Sabouraud's agar, the pH of which is adjusted to 5.0, even at room temperature. This facility of growth permits cultures to be sent to a laboratory at any distance in any season of the year. The growth under such conditions is usually so luxuriant that other organisms are overgrown and the monilia is obtained easily in pure culture. There are undoubtedly many pathogenic types of the fungus, but for practical purposes their differentiation is not essential.

The treatment of monilia vaginitis is usually simple, with prompt relief of symptoms and with complete eradication of the organism, but occasionally is more difficult and time-consuming. In the usual case, the topical application of gentian violet, either in 2 per cent aqueous

solution or in the form of suppositories, will be effective, but in rare instances it will be irritating or quite ineffective, and some other therapeutic agent will be required. Especially in these cases, the combination of potassium iodide and potassium iodate, suggested by Hesseltine, will be useful, and may be employed effectively in any patient with vaginal moniliasis. Alkaline douches may also be employed. Resistant infections occurring during pregnancy are ordinarily relieved by delivery, while those developing in diabetics respond to control of the diabetes with the elimination of the attendant glycosuria.

The active treatment of monilia infections of the vagina during pregnancy is dictated both for the relief of irritation and as a prophylaxis against oral thrush in the newborn. It is well established that many cases of thrush represent birth canal infections, the monilia gaining access to the mouth during parturition. The condition is likewise effectively treated with gentian violet in 1 to 2 per cent aqueous solution.

Undoubtedly, there are many other organisms which are, at least occasionally, responsible for vaginitis, but which have not been recognized as etiologic factors, and for the detection of which, simple laboratory methods have not been devised. It is also probable that they too, like those mentioned, may be present without producing symptoms. This condition, the so-called carrier state, represents one of the present enigmas, that apparently will not be solved until more is known about the physiology of the vagina and its discharge. The occurrence of asymptomatic infections inevitably invokes questions of reduced virulence or of increased local immunity, since either deviation would suffice to explain the clinical anomaly. In gonococcus infections, the evidence points to decreased virulence of the particular strain, whereas in moniliasis the resistance factor is probably involved. In addition, and possibly most important, there is the factor of changing conditions of acidity, of glucose or glycogen deposit, and of the amount of moisture, which may alter the adequacy of the discharge as a medium and thus alter the flora. In prepubertal girls and in post-menopausal women, these factors seem to be under the control of the ovarian hormones, a fact which suggests the possible influence of ovarian products even during the period of sexual maturity and activity.

In conclusion, and from the purely practical standpoint, certain facts must be emphasized.

1. No patient with vaginitis should be treated blindly until after every effort has been made to identify the causative organism by such methods as have been described.

2. After the diagnosis has been made, active therapy should be started according to the preference of the physician, and should be altered if, after fair trial, there is no improvement.

3. The possibility of reinfection must be kept in mind.

4. The patient must be warned to protect others in her family from those infections which are easily transmitted and serious, such as gonorrhea.

5. It must be remembered that mixed infections occur, and that not infrequently an endocervicitis may be responsible for the persistence of leukorrhea even after the specific organisms have been eliminated.

PLACENTA ACCRETA

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PLACENTA accreta is a condition in which the placenta becomes firmly adherent to the uterine wall due to a defective or absent decidua basalis. In this manner, the uterine muscle is exposed to the erosive action of the tropho-blast with a penetration of the muscular wall by the villi. Due to this firm union, the normal mechanism of placental separation is prevented. For normal separation of the placenta, the normal decidual basalis is necessary. After the fetus is expelled, the uterus shrinks in size and thus reduces the size of the placental site. This process causes the placenta to peel off and the formation of small hematomas helps to hasten the separation. The separation occurs in the layer of the decidua basalis which is spongy and easily torn.

Placenta accreta is an extremely rare condition but very definite when one is found. Polak[3] reports that this condition happens in about 1 in

every 6,000 deliveries. Kraul[2] reports three cases in 60,000 deliveries in a period of twenty-three years in Vienna. King,[1] in 1938, checked over the literature and found a total of 109 reported cases. From these and numerous other reports the incidence is about one in 20,000 deliveries. Multipara and primipara are about equally affected.

Etiology.—Inasmuch as placenta accreta is due to an absence of, or pathological condition of, the decidua basalis, one must naturally look for the cause of this irregularity to determine the cause of the firmly adherent placenta. This atrophy may be produced by a too vigorous curettement, or an atrophic endometritis, or a thinning of the endometrium covering a submucous myoma. If the placenta be attached in one of the uterine cornua where the mucosa is thin, one may have a firmly adherent placenta resulting. A mal-functioning corpus luteum must be considered since it is from this structure that the decidua is developed. Finally, in some cases the syncytial cells have more power of erosion and penetration than normal and will penetrate deeply into the uterine wall.

Histologically, the chorion frondosum consists of a connective tissue layer which lies next to the amnion and a layer of villi which are covered by an outer layer of tropho-blastic cells.[4] As each villus goes into the basil decidua which protects the muscular layer, it makes a space by erosion of the mucosa. This space is always larger than the villus which allows a blood space to be formed which is filled with maternal blood. However, villi near the margin of the placenta penetrate more deeply into the uterine wall, thus fastening the two structures together at the periphery. These are known as anchoring villi. However, these are small in number compared to the total of the placental villi and are easily torn off by the contractions of the uterus. In placenta accreta, the uertine wall is extremely thin with a majority of villi perforating into and many even through the muscle.

Microscopically, the basil decidua is either extremely thin or absent and syncytial cells may be demonstrated between the groups of muscle fibers.

Diagnosis.—Placenta accreta must be differentiated from (a) retained separated placenta, (b) simple adhesion of the placenta.

In a retained separated placenta, the normal signs are present, namely, uterine bleeding, descent of the cord, and globular firm uterus. By waiting or attempting the Crede method of separation, this placenta can be expelled. In the simple adhesion, there is no bleeding and none of the other signs are present. The last named, placenta accreta, can only be differentiated by an attempted manual removal. Various authors give the required waiting time at from forty-five minutes to two hours, if there is no excessive bleeding.

In simple adhesion of the placenta, the examining fingers, when introduced into the uterine cavity, readily demonstrate a line of adhesion between the uterine wall and the placenta. This can easily be broken down and the placenta delivered with a minimum amount of trauma. In placenta accreta, no line of adhesion can be found and the placenta cannot be torn loose from the uterine wall. Due to the thinness of the uterine muscles, too forceful an attempt at extraction often results in a perforation of the uterus or severe hemorrhage due to torn muscle fibers or an inversion of the uterus. During any vigorous attempt at removal with placental forceps, small fragments may be torn loose which will result in hemorrhage of various proportions depending upon the size of the placental sinus opened.

Prognosis.—The few cases reported have made the explanation of mortality unreliable. However, in the cases reviewed, a mortality rate of 66.6 per cent was found, due to sepsis, hemorrhage, perforation of the uterus or a combination of these.

Treatment—When a retained placenta is encountered, and none of the usual signs of separation have shown themselves, one must be suspicious of placenta accreta. If it be a simple adhesion of the placenta, removal followed either by packing or ergot is indicated. In placenta accreta, the patient should have immediate supravaginal hysterectomy, since this procedure removes the uterus and the offending placenta and also minimizes danger of infection.

Case History

Mrs. D. E., age 28 years.

Previous history was normal except that two years ago, she had an incomplete abortion at which time she had a curettage done elsewhere. The last normal menstrual period was on April 28, 1938, with an abnormal one-day flow on May 28. Life was felt September 10, 1938. The pregnancy continued normally

until February 10, 1939, at which time the patient demonstrated edema, albuminuria, and a rising blood pressure. It was decided to induce labor and the patient was given castor oil and quinine on the morning of February 11. About 10 A. M., the membranes ruptured

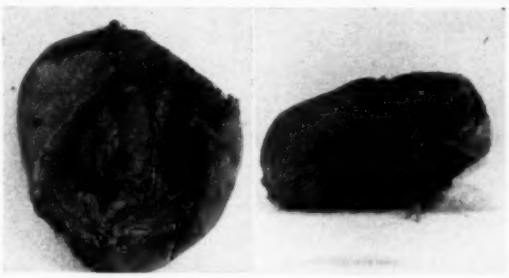

Fig. 1 Fig. 2

Figure 1. Uterus with placenta in situ.
Figure 2. End view showing the extreme thinness of the uterine wall.

with a discharge of a liberal quantity of dark blood-stained, very foul smelling fluid. Pains started at approximately 2 P. M., and labor continued until 12:30 A. M. on February 12, when a normal female infant was delivered with low forceps following a lateral episiotomy. Following the expulsion of the fetus, approximately one gallon of this same blood-stained fluid was discharged. This had an extremely foul odor such as putrified blood. At this time, high up on the cord inside the vagina, a cyst the size of an egg was discovered out of which blood was coming. This was thought to be the cause of the blood leakage into the amniotic fluid. In attempting to clamp the cord above the cyst, the cord was cut with the hemostat. The usual method of extraction of the placenta was tried with no results. After about forty-five minutes wait, with some constant bleeding from the cut part of the cord, it was decided to do a manual removal. This was attempted under ether anesthetic but no line of demarcation between the placenta and the uterine wall could be demonstrated. The placenta was grasped with a placental forceps but only small fragments could be removed. Due to the thinness of the uterine wall and the hemorrhage, only two attempts were made. At this time, the intra-uterine fingers could be felt extremely plain from the outside. A mild degree of shock developed so the uterus was packed tightly and the patient observed the remainder of the night. She quickly came out of shock and that same morning, February 12, at about 11:00 A. M., the blood pressure had risen to 120/70, the pulse was 100 and of good quality. The packing was removed and Crede method attempted but the placenta could not be loosened. She was grouped for transfusion and a supra-vaginal hysterectomy was performed with a minimum amount of blood loss. Immediately following, she was given 500 c.c. of blood by indirect transfusion. The following course was entirely uneventful with the exception of some mild infection and some drainage through the abdominal wall. She was allowed to sit up on the twelfth day and home on the fourteenth day. After going home, she developed a severe pleurisy and considerable vaginal discharge which cleared up entirely on the twenty-eighth day. Since then she has been entirely normal and the baby has progressed extremely well.

Figure 3. Photo-micrograph showing the villi extending into the muscle wall with numerous syncytial cells throughout the muscle fibers.

Pathological Report.—Uterus weighed 2,600 grams and the placenta was attached in the left cornu region. The uterine wall over the placenta was about 1 centimeter in thickness and in other places the wall was 3-4 centimeters. The placenta could only be separated with difficulty and then sections of the wall were torn out and pieces of the placenta remained adherent. The microscopic report was a very thin decidua with syncytial cells throughout the muscle fibers.

Conclusions

1. Placenta accreta is a definite clinical entity although extremely rare.
2. Its etiology is not known but is thought to be an abnormal basil decidua.
3. Diagnosis is made by the inability to separate a retained placenta.
4. Treatment is immediate supra-vaginal hysterectomy followed by transfusion.

References

1. King, P.: Placenta accreta. Ohio State Med. Jour., 34:652, (June) 1938.
2. Kraul, P.: Placenta accreta. Ztschr. f. Gynak., 52:828, 1928.
3. Polak, J. O., and Phelen, Geo. W.: Placenta accreta. Surg., Gyn., and Obst., 38:181, 1924.
4. Schuman, E. A.: Diseases of the embryo and appendages. Curtis: Obstetrics and Gynecology. Philadelphia: W. B. Saunders and Co., 1934. Page 498.

CEREBRAL CALCIFICATION*
(Parkes Weber-Dimitri Type)

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IN VIEW of the frequency with which various forms and types of cerebral calcification are seen on routine roentgenograms of the skull, and in view of the fairly characteristic symptom-complexes which can be produced by pathological cerebral calcification, it is surprising that reports of the condition are not found more frequently in medical literature.

Using Camp's[1] basic outline as a reference, I have made the following classification of cerebral calcification:

I. Physiologic Calcification
   - A. Pineal Gland
   - B. Choroid Plexus
   - C. Falx Cerebri
   - D. Tentorium
   - E. Meninges
   - F. Pacchionian bodies

II. Pathologic Calcification
   - A. Neoplastic Group
     1. Gliomas
     2. Endotheliomas
     3. Aneurysms
     4. Suprasellar cysts
     5. Cholesteatoma
   - B. Non-neoplastic group
     1. Vascular
        - a. Aneurysm
        - b. Hematoma
        - c. Arteriosclerosis with calcification
     2. Old abscesses
     3. Tuberculoma
     4. Parasitic cysts
     5. Encephalitic lesions
        - a. Acute infections such as epidemic encephalitis and malaria
        - b. Intoxications, *i.e.*, CO poisoning
        - c. Various chronic diseases of the brain such as encephalitis interstitialis infantum (Virchow)
        - d. Cerebral atrophy.
     6. Symmetrical calcification
     7. Parkes Weber-Dimitri disease

III. Pseudo-calcification. Deposits simulating calcium in morphology and location, appearing in the brain in three forms (Ostertag)
   - a. Free in tissue as small granules which may become confluent, assuming a mulberry shape.
   - b. In the same form but about the walls of the capillaries, sometimes replacing the capillary wall and obliterating the lumen.
   - c. In the walls of the arteries, appearing as homogeneous or granular masses.

Cerebral calcification may be symmetrical or asymmetrical. Symmetrical calcification is usually found in the basal ganglia and has recently been described by Eaton, Camp and Love.[2] Some authors believe that in this condition we are dealing with pseudo-calcification, but Eaton, et al, proved by chemical analysis in some of their cases that the concretions actually were calcium. Numerous cases have been reported in the literature, and common in all reports are changes in and about the finer cerebral vessels, particularly the lenticular nuclei. According to Eaton, et al, who made a comprehensive review of the literature, most writers described the globules as deeply basophilic staining material lying adjacent to the capillary walls. There is a tendency for the material to coalesce, forming vascular sheaths and concretions. The lumens of the arteries may or may not be constricted. In some cases the dentate nucleus of the cerebellum is involved.

Asymmetrical calcification may occur in any one of the conditions mentioned above under "pathologic calcification." A particularly interesting type is that known as Parkes Weber-Dimitri disease. In this condition, according to Leef[4] the calcifications are unique in that they appear to be tubular and corkscrew-like and seem to follow the outline of the gyri of the brain. At first, the calcifications were thought to be in the blood vessels or in the pia. However, in 1934, Krabbe[3] reported six cases, one of which came to autopsy. Microscopic examination of this material showed that there was no calcification in the pia, but that the deposits were located

*Read before the Minnesota Academy of Medicine, March 8, 1939.

in the cerebral cortex, mostly in the second and third layers. Furthermore, the calcifications bore no relationship to the blood vessels. The etiology of the condition has not been determined.

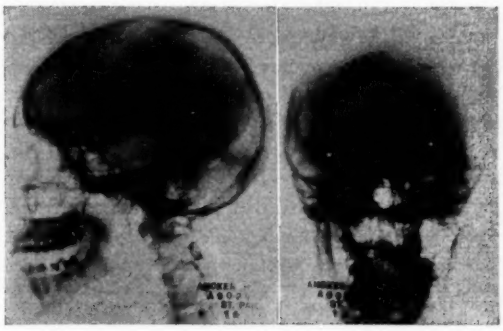

Fig. 1. Fig. 2.

Fig. 1. Lateral view of skull showing deposits of calcium. Note corkscrew-like appearance in the approximate region of the middle cerebral artery.
Fig. 2. Anterior-posterior view showing deposits of calcium, especially on right side.

The reason for the tubular appearance of the calcification is that the thin layers of calcium, being below the molecular and outer granular layers of the cerebral cortex, are separated from each other by these layers. Krabbe has reproduced their appearance on the roentgenogram by taking a brain and coating the gyri with silver nitrate. This salt penetrates a short distance into the cortex and gives a tubular corkscrew-like shadow on the roentgenogram.

Both in symmetrical cerebral calcification and in Parkes Webber-Dimitri disease epileptiform seizures and progressive mental deterioration are the most prominent symptoms. Facial nevi, visual field defects, parkinsonism, cerebral atrophy, and choked disc also have been reported.

Case Report

P. H., a single white female twenty-four years of age, was first seen by me in the out-patient department of the Ancker Hospital in Saint Paul on June 2, 1938. The family history showed that the father, mother, one brother and a sister were alive and well. There were no dead siblings and no family tendencies. The patient was the first child, born normally at full term, after the mother had passed through a normal pregnancy. There were no immediate post-natal complications such as cyanosis or convulsion. Dentition began at seven months and, according to the patient's mother, was normal. Walking began at fourteen months and talking at twenty months. The parents stated that patient seemed to be just like other children her age. During infancy and early childhood there were no serious illnesses, operations or accidents except for a slight head injury without unconsciousness.

The patient's educational history showed fairly normal progress. She started school at the age of seven years and finished the twelfth grade at the age of twenty. Her school records showed that she was a "C" student. This is important in view of the reduced intelligence quotient found after her illness had been present for three years.

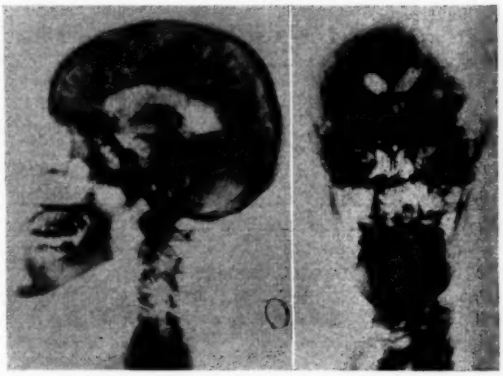

Fig. 3. Fig. 4.

Fig. 3. Lateral view of encephalogram after injection of 140 c.c. of air. Note diffuse cortical atrophy, moderate internal hydrocephalus and also the presence of fluid level.
Fig. 4. Anterior-posterior view of encephalogram after injection of 140 c.c. air. (Same as Fig. 3.) Note marked cortical atrophy especially on the right.

Relevant points in the past history were an appendectomy and cholecystectomy in 1933 because of vague abdominal pains. In 1935 the patient was in another hospital where a diagnosis was made of renal lithiasis. However, the stone never was found and the patient was said to have passed it.

The present illness began in 1935 when the patient began to have attacks of petit mal. These were quite typical and continued at irregular intervals for several months. Then grand mal attacks appeared. She would have a psychic aura during which she became momentarily confused. Then she suddenly would lose consciousness and fall to the ground in a generalized convulsion, during which she would chew her tongue and pass her urine. There never were any focal attacks observed. Between the attacks of grand mal, which came on from once a week to once in three weeks, patient would have attacks of severe lancinating pain in various parts of her body and extremities. These pains were colicky in nature and might well lead one to suspect gallbladder or renal colic.

The neurological examination showed the pupils to be dilated, but equal and regular and responding to light and in accommodation. Extraocular movements were normal but there was a divergent squint due to a weakness of the right internal rectus. There was bilateral optic atrophy and examination of the visual

fields showed a left homonymous partial hemianopsia. The cranial nerves were otherwise normal. The extremities showed normal muscle volume, tone, strength, and coördination. All tendon and superficial reflexes were present and equal and no pathological reflexes were elicited. Sensation was normal in all modalities.

The blood Wassermann was negative and the cerebrospinal fluid was clear, under normal pressure, contained 4 lymphocytes, .045 gm. protein, 720 mg. chloride per 100 c.c., gave a negative Wassermann reaction, and the colloidal gold curve read 0001110000. Blood calcium was 9.5 mg. and 11.5 mg. per 100 c.c.

The patient was admitted to the hospital for encephalography and further study. Upon entering the hospital she complained bitterly of sharp, lancinating pains throughout her body, arms, and legs. On the second day she developed an acute psychotic episode and mechanical restraint was required. For three days she was noisy, incoherent, and shouted that she was being tortured. The excitement subsided without any special treatment and further studies could be carried out.

The I.Q. (Kuhlman's) was 62 per cent. X-rays of the skull showed characteristic deposits of calcium distributed asymmetrically in the cerebral cortex, principally in the right cerebral hemisphere. One area had a typical tubular corkscrew appearance (Fig. 1).

Encephalography after injection of 135 c.c. of air showed good filling of the ventricular system, including the cortical fluid pathways. The cortical markings on the left half of the brain were less numerous than average, making it necessary to consider the possibility of arachnoiditis on the left side. The ventricles showed about normal size and configuration except for slight enlargement of the left lateral ventricle, especially posteriorly, and displaced to the left. This was associated with considerable contraction of the brain as if due to atrophy. The subdural space was obliterated superiorly over the brain surface on the left. This study suggested left brain atrophy with evidence also of arachnoiditis bilaterally.

After the encephalogram the patient seemed to be much brighter and she complained much less of pain. Two weeks after admission she was discharged from the hospital and followed in the out-patient department, where she has since been reporting once a month. In January, 1939, the grand mal seizures reappeared and were not controlled by the administration of 6 grains of phenobarbital daily. Accordingly she was placed on dilantin, 1½ grains four times a day, and she has been free from seizures since March 23, 1939. Her psychiatric status has not altered. She continues to be dull, irritable, and the parents find her a problem in discipline. Recently she has become intensely erotic and without close supervision probably would become sex delinquent. The optic atrophy has not progressed and the visual fields have not altered.

Comment

The configuration of the calcium deposits as shown by the roentgenogram in this case correspond closely to that described in Parkes Weber-Dimitri disease. The etiology of this condition is unknown, but at least in this patient the condition is an acquired one. With normal birth and developmental history, a fairly normal scholastic record, and the absence of any early personality changes, it is difficult to see how the condition could have been congenital. Whether the cerebral atrophy preceded or followed the process of calcification is impossible to say. It is conceivable that there was at one time an internal hydrocephalus. This could account for the optic atrophy and possibly also the dilated ventricle. The absence of a history of headache does not rule out an early chronic hydrocephalus. I recently saw a case of advanced internal hydrocephalus with bilateral consecutive optic atrophy which at autopsy proved to be due to an enormous pituitary adenoma which had grown up out of the sella encroaching on the floor of the third ventricle. This patient never had complained of headache or any other symptoms of increased intracranial pressure.

It is interesting to note that in this case some of the calcium deposits were in the right parieto-occipital area. Interference with the optic radiations in this region would explain the left homonymous partial hemianopsia.

Summary

1. A working classification of physiological and pathological cerebral calcification is presented.

2. Pathological cerebral calcification frequently results in epileptic convulsions and mental deficiency. Facial nevi, cerebral atrophy, visual field defects, and optic atrophy also may occur.

3. Parkes Weber-Dimitri disease is a peculiar type of cerebral calcification in which the concretions have a characteristic roentgenographic appearance.

4. A case of Parkes Weber-Dimitri disease is reported.

References

1. Camp, J. D.: Intracranial calcification and its roentgenologic significance. Am. Jour. Roentgenol., 23:615-624, 1930.
2. Eaton, L. McK., Camp, J. D., Love, J. G.: Symmetrical cerebral calcification, particularly of the basal ganglia, demonstrable roentgenographically, and calcification of the finer cerebral blood vessels. Arch. Neurol. and Psych., 41:921-942, (May) 1939.
3. Krabbe, K. H.: Facial and meningeal angiomatosis associated with calcifications of brain cortex; clinical and anatomopathologic contribution. Archiv. Neurol. and Psych., 32:737-755, 1934.
4. Leef, E.: Epilepsy associated with cerebral calcification. Radiology, 27:370, 1936.
5. Ostertag: Virchow's Archiv. f. Path. Anat., 275:828, 1930 (quoted by Eaton).

PARENTERAL ADMINISTRATION OF FLUIDS: PRINCIPLES AND INDICATIONS IN SURGICAL TREATMENT*

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MORE and more it is being realized that the successful practice of surgery is dependent, in many instances, on a careful study of the metabolism of the patient in all stages of his illness. Attention must be paid to the requirements of water and electrolytes, especially in cases in which the normal mechanisms of exchange of fluid are interfered with by disease or by surgical procedures. These requirements of water and electrolytes are both quantitative and qualitative, for harm may be done either by inadequate or excessive exhibition of fluids or by the employment of unsuitable solutions. Because the oral and rectal routes of administration of fluid are so often incapable of adequately meeting the needs of surgical patients, particular reference will be made to the parenteral routes and to the solutions which may be given by such routes.

Parenteral administration of fluids, at the present time, is a relatively safe and certain procedure. Its origin and its development have been well outlined by Keith.[27] Thomas Latta[31] may be considered the father of this method of treatment for, in the Edinburgh epidemic of malignant cholera, in 1831 and 1832, he introduced the practice of "the copious injection of aqueous and saline fluid into the veins" of patients in a state of collapse. He did this because the chemists had told him that the blood was deficient in these materials and he found that temporary benefit and even recovery sometimes followed. However, it was not until the World War, when "gum-saline"† solutions were introduced by Bayliss,[3] by Drummond and Taylor,[12] and by Keith[28] that the method found widespread application. The demonstration of pyrogenic substances in certain distilled waters by Seibert[42] led to greater appreciation of the need for meticulous care in the preparation of solutions, whereas the introduction of the method of intraperitoneal injection of saline solutions of Blackfan and Maxcy[5] and of the continuous intravenous "drip" by Matas[33] improved the methods of administration. In the last decade, considerable work has been done on the metabolic requirements of surgical patients, particularly for water and electrolytes, and the composition of the fluids needed to satisfy these requirements has become better understood.

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†The solution used consisted of 6 per cent acacia in physiologic saline solution.

The Requirements for Water

Maddock and Coller[32] have recently written an excellent review of this problem and they point out that an individual, whether healthy or sick, must take sufficient fluid in order to ensure that his body has at least an adequate amount of water with which to carry out the normal physiologic activities. They found that a normal individual, who is not sweating unduly, loses each day 600 to 1500 c.c. of water in the urine, 150 to 200 c.c. in the stools and 1000 to 1500 c.c. in vaporization from the skin and lungs. Thus, 1750 to 3200 c.c. of fluid are required daily in order to maintain a proper water balance. This amount need not all be taken as water to drink because a considerable proportion of it can be derived from the water content of the food and, to a lesser extent, from the combustion of the constituent principles of food. It is estimated that, in this way, a routine maintenance diet will yield 1000 to 1500 c.c. of water daily. Even in starvation 500 c.c. of water become available daily from the materials of the body used for energy.

On the other hand, a sick patient may have requirements for water that are different from those considered adequate for a normal individual and, in estimating the probable amounts required, the nature of his illness must be considered. Among surgical patients, as among normal individuals, the excretion of water serves two main functions: the elimination of waste materials in the urine and the dissipation of heat from the body by sweat and respiration. (The loss of water in the normal stool is insignificant and can be disregarded.) The vaporizing pro-

cesses have first claim on the available water, whereas the kidneys function with what is left and, therefore, it is necessary that the patient receive sufficient water to enable the kidneys to excrete an adequate amount of urine each day. Lashmet and Newburgh[30] have shown that about 35 gm. of waste material are excreted in the urine daily and that, when the kidney can concentrate urine to a specific gravity of 1.032 to 1.029, this amount may be eliminated in 500 c.c. of urine; whereas, when the kidneys can concentrate urine only to 1.014 or 1.010, nearly 1500 c.c. of urine are required for such elimination. This means that a daily output of 1500 c.c. of urine should be aimed at for the average surgical patient in order to allow a reasonable margin of safety against the retention of waste materials.

In assessing the amount of water required for vaporization it must be remembered that, although a normal individual may subsist on an allowance of 1000 to 1500 c.c. daily, surgical patients frequently require more. Patients who have fever and are actively sweating vaporize 1500 to 2500 c.c. daily, whereas patients who have exophthalmic goiter vaporize 1500 to 2000 c.c.[32] Maddock and Coller think that 2000 c.c. of water per day is a safe provision for a surgical patient. This figure together with an allowance of 1500 c.c. of water for urine leads to an estimate of 3500 c.c. for the daily requirements of water for the average surgical patient.

Even this requirement of 3500 c.c. daily is not sufficient in some cases. Maddock and Coller[32] and Bingham[4] have drawn attention to the great loss of fluid from the body which occurs during surgical operations and during the first twenty-four hours afterward. Although the loss of blood which may occur from hemorrhage during the course of routine operations is often greater than the surgeon suspects,[18, 32] much the greatest source of this loss of fluid is through vaporization, which may account for as much as 2600 c.c. of water.[4] Again, in certain conditions of the gastro-intestinal tract, such as vomiting from any cause, intestinal fistula, and severe diarrhea, large quantities of water may be lost in addition to that lost by the usual routes. The harmful effects that follow these abnormal losses may be prevented by the administration of amounts of fluid equal in volume to that of the abnormal amounts lost, in addition to the provisional allowance of 3500 c.c. a day. If this is not done, serious symptoms of dehydration will ensue when water equal in amount to 6 per cent of the body weight has been lost from the body[32] and azotemia, acidosis, and oliguria will manifest themselves.

The figures which have been considered apply only to adults; the data that apply to children in this respect are much less certain. However, the rule still holds that water should be given until an adequate daily urinary output is established. Table I indicates the normal urinary output among children, according to Campbell.[7]

TABLE I. AVERAGE OUTPUT OF URINE AMONG CHILDREN IN TWENTY-FOUR HOURS (CAMPBELL)

Age	Output in urine, c.c.
10 days to 2 months	250- 450
2 months to 1 year	400- 500
1 to 3 years	500- 600
3 to 5 years	600- 700
5 to 8 years	650-1000
8 to 16 years	800-1600

Requirements for Electrolytes

Sodium chloride is the principal salt required for the maintenance of normal activity, the body possessing normally 150 to 200 gm.[43] of this salt, of which about 30 gm. are in the blood. There are wide variations in the amounts of sodium chloride that normal subjects can take. It has been shown that the basal requirements are in the neighborhood of 1 to 2 gm. daily,[13] whereas the continued ingestion of more than 30 to 40 gm. daily by normal subjects leads to edema.[2] Among patients who have hypoproteinemia[26] consequent to inanition, to chronic sepsis, and to the wasting diseases, even smaller quantities of salt will lead to retention of fluid. On the whole, a daily intake of 8 to 12 gm., which is the amount present in an average diet, is a satisfactory amount to be given to surgical patients who do not have an initial low concentration of plasma chlorides.

When hypochloremia is present, larger amounts of sodium chloride are necessary. The surgical conditions which frequently lead to this state of metabolic upset are conditions in which there are abnormal losses of[36] the digestive secretions (profuse and persistent vomiting from any cause, prolonged aspiration of the stomach or duodenum, high intestinal fistulæ, and severe

diarrhea), peritonitis,[38] and severe burns;[10] rarely does sweating alone lead to depletion of salt in surgical conditions because the concentration of sodium chloride of sweat is low (0.2 per cent).[35] Salt depletion as the result of abnormal losses of chloride in the gastro-intestinal secretions may be prevented by the administration of physiologic saline solution in amounts equal to the volume of the secretions lost. The concentration of sodium chloride in the digestive secretions rarely exceeds 0.6 per cent and therefore loss of chloride by this route is amply covered by physiologic saline. Lyall and I[13] have shown that, if hypochloremia should develop, sodium chloride is required in amounts of 15 to 25 gm. for each 100 mg. the plasma chloride level is below normal. The normal concentration of plasma chloride is 540 to 620 mg. per 100 c.c. Care should be taken that not too much salt is given lest edema of the lungs and extremities results.

Osterberg,[15] Bargen and I have recently drawn attention to the need for salts of potassium and calcium in small quantities as well as sodium chloride. We pointed out that the requirements for these salts are probably amply met by the provision of Ringer's solution instead of plain saline solutions. Ringer's solution has the additional advantage that it is less likely to lead to edema and retention of water[15, 32] than plain saline solutions.

In many surgical conditions, interference with the acid-base equilibrium occurs. Many excellent reviews of this problem have appeared, among which may be mentioned those of Van Slyke[48] and Hartmann.[21] Alkalosis may be attributable to increased "alkali reserve," as following the ingestion of large quantities of alkali by susceptible patients during the alkaline treatment of peptic ulcer or, more frequently, to loss of chloride ions as in cases of severe vomiting and high intestinal fistula. The former condition will clear up following the cessation of administration of alkali and the provision of an adequate intake of fluid; the latter condition usually will respond to the administration of saline solutions, the excess sodium ions being excreted in the urine. Very rarely are other measures required. In severe cases of alkalosis, as when the carbon dioxide combining power is more than 100 vols. per cent, calcium may be required to control tetanic manifestations.

Acidosis is a more serious condition than alkalosis and may be caused by a great loss of the bases of the plasma as in cases of severe diarrhea, by the retention of nonvolatile acid ions, such as phosphates and sulfates in cases of renal failure, or by the presence of abnormal acids such as the ketone bodies in cases of diabetic coma, dehydration, and certain cases of intestinal obstruction.[14] Because hypochloremia, dehydration, and ketosis are very frequent accompaniments of acidosis, the standard treatment is the administration of solutions of sodium chloride and d-glucose. In severe cases, as when the carbon dioxide combining power is less than 30 vols. per cent, solutions containing alkaline salts are required in addition.

Requirements for Nutrient Materials

Solutions of d-glucose (dextrose) can be used with advantage to combat ketosis and to spare body protein. One thousand cubic centimeters of a 5 per cent solution of d-glucose can be taken intravenously in an hour without sugar appearing in the urine.[49] However, such solutions cannot be used with practicability to provide all the energy required by the body, which, in the case of a man weighing 70 kg., is about 2100 calories daily and which theoretically would require that a 10 per cent solution of d-glucose be given parenterally in amounts of about 5000 c.c. for its satisfaction. Therefore, if a patient is in urgent need of nourishment, but cannot take any or sufficient food by mouth, feeding by gastric or duodenal intubation, by gastrostomy or by jejunostomy should be considered.

Solutions Available for Parenteral Administration

The following solutions have received recognition in parenteral fluid therapy.

Solutions of sodium chloride.—Sodium chloride is used in varying concentrations. Physiologic saline varies from 0.85 to 0.90 per cent (isotonic) although, clinically, 1 per cent solutions are often considered in this category. Hypertonic solutions of 10 to 30 per cent are used intravenously as dehydrating agents and for stimulation of the bowel, whereas solutions of 3 to 5 per cent are used by some authorities for the rapid correction of hypochloremia.

Solutions of d-glucose.—These solutions are used either in isotonic (5 per cent) or in hyper-

tonic (10 per cent) concentration, depending on requirements for energy and the degree of dehydration present. Solutions of d-glucose (dextrose) and sodium chloride are often combined and probably a 5 per cent solution of d-glucose in physiologic saline is the solution most widely used in North America.[9]

Modified solutions of sodium chloride.—Ringer's solution. An isotonic solution would contain sodium chloride 0.8 per cent, potassium chloride 0.05 per cent and calcium chloride 0.03 per cent. Hartmann[22] recommended a hypotonic solution of sodium chloride, 0.60 per cent; potassium chloride, 0.04 per cent; calcium chloride, 0.02 per cent and magnesium chloride, 0.02 per cent. Both of these solutions are improved by combination with a 5 per cent solution of d-glucose.[24]

Hartmann's solution.—Hartmann recommended the following formula: sodium chloride, 0.60 per cent; potassium chloride, 0.04 per cent; calcium chloride, 0.02 per cent; magnesium chloride, 0.02 per cent; and sodium lactate, 0.30 per cent.

Solutions of acacia.—The solution customarily used contains 6 per cent acacia in physiologic saline (gum saline).

Solutions of sodium lactate and sodium bicarbonate.—Isotonic solutions of sodium lactate (1.8 per cent) or sodium bicarbonate (1.3 per cent) are used intravenously in the treatment of severe acidosis. Sodium bicarbonate is also used in 5 per cent solution.

Solutions of ammonium chloride.—Ammonium chloride in a concentration of 0.5 per cent may be used occasionally in the treatment of alkalosis.

Hartmann[22] and Thompson[47] have given instructions for the preparation of these solutions. In the prevention of untoward reactions following intravenous infusion, Thompson[47] and Rademaker[39] stressed the importance of using pure chemical and pyrogen-free distilled water, which can be obtained by triple distillation, by the use of a baffle plate still, or by filtration through a 200 second Zsigmondy filter.[8] Only tubing made of pure gum rubber and hard nonsoluble glassware should be allowed in the apparatus used in the administration of solutions and these should be thoroughly boiled and cleansed before initial use.

Routes of Administration

Fluids may be given by three different parenteral routes, namely the intravenous, the subcutaneous and the intraperitoneal routes. The use of each of these routes has its advocates. The administration of fluids by one or more of these routes is indicated whenever the patient is unable to ingest and absorb sufficient fluid by mouth to satisfy his immediate requirements. Although rectal administration gives satisfactory results when small quantities are indicated and for short periods, the parenteral routes have great advantages when large quantities are required either rapidly or over a long period of time. The factors governing the choice of method in the individual case may be summed up as follows: For the administration of large quantities of isotonic solution, any one of the three parenteral routes may be used. Saline solutions usually can be administered satisfactorily into the loose tissues of the axilla, the outer aspects of the thigh and under the breast in quantities of 2,000 to 3,000 c.c. daily. After a few days, however, these routes become unbearably painful. Among infants, the intraperitoneal route, as introduced by Blackfan and Maxcy,[5] is on the whole satisfactory, although, according to Ravenel, it is not entirely free from risk.[40] The intravenous route, popularized by Matas, with the intravenous drip[33] is satisfactory for all solutions, isotonic and hypertonic; but very strict care must be taken in the preparation of the apparatus and solutions in order to prevent the occurrence of a reaction. Special solutions, such as those containing acacia, sodium bicarbonate, sodium lactate, and ammonium chloride, should be given only by the intravenous route.

Recently, it has been shown, both clinically[17,45] and experimentally,[41] that the continuous intravenous "drip" as usually practiced may lead to the occurrence of pulmonary embolism secondary to thrombosis set up in the irritated vein. This has given rise to the "intermittent drip" as suggested by Friedrich and Buchaly.[17] Fluids are given by drip for short periods only, about 1,000 to 2,000 c.c. being given at the rate of a litre in one or two hours, and additional quantities, if required, being given later in the day. Isotonic solutions alone are employed and a different vein is selected each time, the fluid being infused by a needle and not by a cannula tied into a vein. With reasonable skill it is possible to in-

troduce a needle into a vein of the leg or arm, after a preliminary cutaneous wheal has been raised with novocain at the site of puncture. If the veins, at first sight, appear collapsed, they usually can be dilated by the application of hot packs to the limb for about twenty minutes.

I shall now discuss the indications for use of the various solutions. Excellent papers on this subject have been furnished by Keith,[27] Hartmann,[22] Stewart[44] and Nadler.[36]

Indications for the Administration of Solutions of Saline and/or d-Glucose Parenterally

The administration of fluids parenterally is indicated whenever the patient is unable to satisfy adequately his immediate requirements for fluid by ingestion, or by enterostomy, if such already has been established. When large quantities of fluid must be given, infusions administered parenterally are preferable to infusions administered rectally because of the greater certainty of their absorption and utilization. The extent of their use is governed by two considerations: (1) the state of the daily water balance as determined by a carefully kept chart contrasting the total daily intake and output of fluids, and (2) the state of the electrolytes of the blood as shown by determinations of the plasma chlorides (normal 540 to 620 mg. per 100 c.c. of plasma), the carbon dioxide combining power of the plasma (normal 55 to 65 volumes per cent of plasma) and blood urea (normal 20 to 35 mg. per 100 c.c. of blood) made, at least, on each alternate day.

The first thing to be considered is the requirement for water. As mentioned earlier, the average surgical patient requires an intake of about 3,500 c.c. daily in order to insure an adequate urinary output of about 1,500 c.c. of urine; a patient suffering from established dehydration or abnormal losses of fluid may require 5,000 c.c. or even more. On the chart attention should be paid to the amount of urine passed each day and to the difference between the total intake of fluid and the total output of fluid. The key to the correction of dehydration is the presence of an adequate output of urine and this is a signal for cutting the intake of fluid down to maintenance levels. Once an adequate urinary output has been established, a difference of more than 1,500 to 2,000 c.c. between the total intake of fluid and the total output of fluid signifies retention of water and steps should be taken to restrict the intake. Edema, however, is more the result of retention of salt than of excessive intake of fluid[32] and the patient should be examined daily for its earliest manifestations.

Cabot and Iber[6] have pointed out that a surgeon who is quick to recognize the signs of dehydration when a patient is seen for the first time may be slow to recognize the same signs if a patient is being seen every day, especially if the patient is receiving apparently a good supply of water, but actually is becoming dehydrated because of abnormal losses of fluid. The resulting poor output of urine is attributed erroneously to a reflex or toxic suppression of renal function, whereas the real fact is that the patient has not been given sufficient water for the formation of urine. If the fluid balance had been carefully charted, such a state of affairs would not have materialized.

As long as the concentration of plasma chloride is normal (540 to 620 mg. per 100 c.c. of plasma), sodium chloride should not be given in amounts larger than 6 to 10 gm. daily unless there is an abnormal loss of gastro-intestinal secretions as in vomiting, gastric aspiration, intestinal fistula, or diarrhea, when additional physiologic saline in amounts equal to the abnormal volume of fluid lost should be given (the average concentration of sodium chloride in the various gastro-intestinal secretions is about 0.5 to 0.6 per cent). An intake of larger amounts of salt invites the occurrence of retention of water and edema. The salt required may be given as physiologic saline or Ringer's solution with or without d-glucose and, if additional fluid is then needed, a 5 per cent solution of d-glucose may be given. If hypochloremia is present, additional amounts of a solution of sodium chloride are indicated. Many workers have advocated the use of 3 to 5 per cent solutions, but Lyall and I[14] are of the opinion that better results follow the use of physiologic solutions which incidentally correct and do not aggravate the dehydration. We have shown that, roughly, 15 to 25 gm. of sodium chloride are required to raise the lowered concentration of plasma chloride 100 mg. per cent. It is significant that Orr,[37] who, in association with Haden,[19] introduced the use of very large quantities (1 gm. per kilogram of the patient's body weight on the first day) of sodium chloride in the treatment of intestinal obstruction, has, since that time, modified his views be-

cause such treatment produces a tendency toward edema.[37]

Recently Osterberg, Bargen and I[15] have drawn attention to the needs of the body for small quantities of calcium and potassium in addition to sodium. We reported three cases of intestinal obstruction in which the additional administration of salts containing these metals was of value and we favor the routine use of Ringer's solution in preference to solutions of sodium chloride.

When saline solutions are being given intravenously, it is well to combine d-glucose in 5 per cent solution in order to prevent ketosis and to spare the proteins of the body.

Indications for the Administration of Solutions of Acacia and Saline

The use of acacia (5 per cent in Locke's solution) was first introduced in 1917 by Hurwitz in the United States[25] and by members of the Special Investigative Committee of the Medical Research Council[3,12,28] (6 per cent acacia in physiologic saline solution) for the treatment of cases of surgical shock. One of the principal features of this condition is a diminution in the volume of the circulating blood. In the production of this diminished volume, the principal factor known appears to be a reduction in the total volume of the plasma, the result of permeation of plasma-like fluids through the vascular walls.[20,28,34] As a result, actual reduction of the total volume of the blood occurs simultaneously with an increase in the concentration of the cellular elements of the blood. In cases which end fatally, death results from circulatory failure brought about by the presence of an insufficient amount of circulating blood.

One of the principal objectives of the treatment of shock, therefore, is to restore the volume of the circulating blood in order to maintain the circulation. The most direct method is to increase the volume of the blood by the intravenous administration of fluids. Transfusions of blood have been tried and found satisfactory, but unless the patient has had an extensive hemorrhage previously or is anemic, transfusions are unnecessary because what the patient requires is fluid to increase the volume of blood, not corpuscles, of which he already has an ample supply. Theoretically, transfusion of blood plasma would be the ideal method of therapy. Although several workers have considered its advantages, few appear to have tried it. Filatov and Kartasevskij[16] have reported the successful transfusion of plasma in amounts of 300 to 350 c.c. in two cases of shock. They used blood plasma of group AB (universal recipient), which does not agglutinate the red cells of any other blood group, so that preliminary blood grouping of their patients was not necessary. Amberson[1] has pointed out in a review of the problem of substitutes for blood that plasma is to be preferred to serum because of the formation, in the latter, of vasodilator and constrictor substances produced in the act of clotting.

Acacia in physiologic saline solution is an effective substitute for blood plasma[1] and is probably still the most useful agent that we possess for the treatment of surgical shock. The presence of acacia (6 per cent) in the solution raises the colloidal osmotic pressure to a level equivalent to that of plasma, thereby preventing the rapid elimination of electrolytes and water from the circulating blood by excretion through the kidney and permeation through the capillary walls which occurs when simple saline solutions are used alone. In conditions of shock, the solution of acacia and saline should be given slowly by intravenous drip in amounts of 500 to 1,000 c.c. Zunz and Govaerts[50] have shown that, when shock is present, too rapid administration of blood or fluids intravenously may cause the circulation to falter.

The majority of workers have found the intravenous administration of acacia (6 per cent) in physiologic saline solution to be a relatively safe and satisfactory procedure. A solution of acacia is apparently innocuous when introduced into the blood stream, passes within a short time into the tissues and is slowly mobilized and excreted in the urine. Keith, Power and Wakefield[29] have demonstrated its presence in the blood of patients three years after administration without any apparent detrimental effects having been manifested. The repeated daily administration of large amounts of acacia in saline solution, as advocated by Hartmann and his associates[23] in the treatment of nephrotic edema may lead, in some cases, to hepatic damage, but in the amounts used in the treatment of shock, this scarcely seems possible.[1] The recent warning of Studdiford[46] as to this possibility would seem to be the result of the use of solutions which had not been properly prepared.

Indications for the Administration of Solutions of Sodium Bicarbonate and/or Sodium Lactate

These solutions are used in the correction of severe degrees of acidosis such as are encountered in severe cases of diarrhea, dehydration, diabetic coma and renal failure. As mentioned earlier, in cases of minor degrees of acidosis, chief reliance should be placed on the administration of solutions of saline and d-glucose to correct dehydration, supply electrolytes and combat ketosis. Hartmann's solution may be used instead and has the advantage that it contains sodium lactate in small quantities. On the other hand, when serious degrees of acidosis are present, as when the carbon dioxide combining power of the plasma is less than 35 vols. per cent, more concentrated solutions of sodium bicarbonate or sodium lactate are used. Sodium bicarbonate may be used in 5 per cent solution intravenously, 5 to 10 c.c. per kilogram of body weight being given.[22,44] Isotonic solutions of sodium bicarbonate (1.3 per cent) or sodium lactate (1.8 per cent) are preferable and are given in amounts of 10 to 20 c.c. per kilogram of body weight. Van Slyke gives an excellent line table[48] by which the probable amounts required may be estimated. I have found it very important to control the administration of these solutions by frequently determining the carbon dioxide combining power of the plasma, because sometimes a condition of acidosis changes readily to that of alkalosis.

Indications for the Administration of Solutions of ammonium chloride

The majority of conditions of alkalosis associated with azotemia and hypochloremia will clear up following the correction of the hypochloremia by means of administration of solutions of sodium chloride or Ringer's solution. Although it may take some time for the concentration of plasma chloride to become normal, I have not seen harm follow operation in cases in which the carbon dioxide combining power of the plasma was in the region of 80 vols. per cent at the time of operation, provided that the values for blood urea and plasma chloride were within normal limits. In the usual case[11] in which the carbon dioxide combining power of the plasma remains in the region of 100 vols. per cent in spite of treatment along accepted lines, injections of ammonium chloride (0.5 per cent) in amounts up to 1,000 c.c. may be given. The administration should be controlled by determining the carbon dioxide combining power of the plasma frequently.

Summary

Parenteral administration of fluid is now a well established, safe and convenient method of introducing water and electrolytes into the body. It is indicated whenever the patient is unable adequately to satisfy his immediate requirements for these substances by ingestion through the natural channel. However, before it can be used to best advantage, the surgeon must know these immediate requirements and the potentialities of the solutions available to him.

This paper discusses the physiologic requirements of surgical patients for water and electrolytes; it enumerates the solutions available for parenteral use for the satisfaction of these requirements and it outlines the principles on which the use of these solutions is based.

References

1. Amberson, W. R.: Blood substitutes. Biol. Rev. of Cambridge Philosophical Soc., 12:48-86, (Jan.) 1937.
2. Baird, M. M., and Haldane, J. B. S.: Salt and water elimination in man. Jour. Physiol., 56:259-262, (May 16) 1922.
3. Bayliss, W. M.: Intravenous injections to replace blood. National Health Insurance, Med. Res. Com., Special Report No. 25, London, H. M. Stat. Office, 1919, pp. 11-41.
4. Bingham, D. L. C.: The water requirements of surgical patients. Brit. Med. Jour., 1:67-70, (Jan. 8) 1938.
5. Blackfan, K. D., and Maxcy, K. F.: The intraperitoneal injection of saline solution. Am. Jour. Dis. Child., 15:19-28, (Jan.) 1918.
6. Cabot, Hugh, and Iber, F. C.: Anuria. Proc. Staff Meet., Mayo Clin., 8:354-359, (June 7) 1933.
7. Campbell, M. F.: Pediatric Urology. New York: The Macmillan Company, 1937, vol. 1, p. 36.
8. Co Tui, McCloskey, K. L., Schrift, Milton, and Yates, A. L.: A new method of preparing infusion fluids: based on removal of pyrogen by filtration. Jour. A.M.A., 109: 250-252, (July 24) 1937.
9. Cutter, R. K.: The use of large volume intravenous injections. Jour. A.M.A., 106:1250-1252, (Apr. 11) 1936.
10. Davidson, E. C.: Sodium chloride metabolism in cutaneous burns and its possible significance for a rational therapy. Arch. Surg., 13:262-277, (Aug.) 1926.
11. Deeds, Douglas: Severe pyloric obstruction: report of a case. Proc. Staff Meet., Mayo Clin., 13:456-460, (July 20) 1938.
12. Drummond, Hamilton, and Taylor, E. S.: The use of intravenous injections of gum acacia in surgical shock. National Health Insurance, Med. Res. Com., Special Report No. 25, London, H. M. Stat. Office, 1919, pp. 135-137.
13. Falconer, M. A., and Lyall, A.: Requirements of sodium chloride. Brit. Med. Jour., 2:1116-1118, (Dec. 4) 1937.
14. Falconer, M. A., and Lyall, A.: Blood chemistry in intestinal obstruction: changes in response to treatment. Lancet 2:1472-1477, (Dec. 25) 1937.
15. Falconer, M. A.: Osterberg, A. E., and Bargen, J. A.: (Unpublished data).
16. Filatov, A., and Kartasevskij, N.: Die Transfusion von menschlichem Blutplasma als blutstillendes Mittel. Zentralbl. f. Chir., 62:441-445, (Feb. 23) 1935.
17. Friedrich, H., and Buchaly, J. F.: Ist die intravenöse Dauertropfinfusion mit Gefahren verbunden? Zentralbl. f. Chir., 59:131-137, (Jan. 16) 1932.
18. Gatch, W. D., and Little, W. D.: Amount of blood lost during some of the more common operations. Jour. A.M.A., 83:1075-1076, (Oct. 4) 1924.
19. Haden, R. L., and Orr, T. G.: Use of sodium chloride in treatment of intestinal obstruction. Jour. A.M.A., 82:1515-1517, (May 10) 1924.
20. Harkins, H. N., and Harmon, P. H.: Plasma exudation:

loss of plasma-like fluid in various conditions resembling surgical shock: experimental study. Ann. Surg., 106: 1070-1083, (Dec.) 1937.
21. Hartmann, A. F.: Prevention and treatment of severe disturbances in water and electrolyte balance. Jour. Iowa M. Soc., 27:451-457, (Sept.) 1937.
22. Hartmann, A. F.: Theory and practice of parenteral fluid administration. Jour. A.M.A., 103:1349-1354, (Nov. 3) 1934.
23. Hartmann, A. F., Senn, M. J. E., Nelson, Martha V., and Perley, A. M.: The use of acacia in the treatment of edema. Jour. A.M.A., 100:251-254, (Jan. 28) 1933.
24. Horsley, J. S.: The intravenous administration of dextrose in Ringer's solution: with particular reference to its use in acute abdominal conditions. Ann. Surg., 98: 678-684, (Oct.) 1933.
25. Hurwitz, S. H.: Acacia solutions. Jour. A.M.A., 68:699, (Mar. 3) 1917.
26. Jones, C. M., and Eaton, F. B.: Postoperative nutritional edema. Arch. Surg., 27:159-177, (July) 1933.
27. Keith, N. M.: Intravenous medication: physiologic principles and therapeutic applications. Jour. A.M.A., 93:1517-1522, (Nov. 16) 1929.
28. Keith, N. M.: Blood volume changes in wound shock and primary hemorrhage. National Health Insurance, Med. Res. Com., Special Report No. 27, London, H. M. Stat. Office, 1919, 16 pp.
29. Keith, N. M., Power, M. H., and Wakefield, E. G.: The detection and persistence of acacia in the blood. Proc. Staff Meet., Mayo Clin., 10:38-41, (Jan. 16) 1935.
30. Lashmet, F. H., and Newburgh, L. H.: Comparative study of excretion of water and solids by normal and abnormal kidneys. Jour. Clin. Investigation, 11:1003-1009, (Sept.) 1932.
31. Latta, Thomas: Malignant cholera: relative to the treatment of cholera by the copious injection of aqueous and saline fluids into the veins. Lancet, 2:274-276, (June 2) 1831-1832.
32. Maddock, W. G., and Coller, F. A.: Water balance in surgery. Jour. A.M.A., 108:1-6, (Jan. 2) 1937.
33. Matas, Rudolf: The continued intravenous "drip"; with remarks on the value of continued gastric drainage and irrigation by nasal intubation with a gastroduodenal tube (Jutte) in surgical practice. Ann. Surg., 79:643-661, (May) 1924.
34. Moon, V. H.: The shock syndrome in medicine and surgery. Ann. Int. Med., 8:1633-1648 (June) 1935.
35. Moss, K. N.: Some effects of high air temperatures and muscular exertion upon colliers. Proc. Roy. Soc., London, s.B., 95:181-200, (Aug. 1) 1923.
36. Nadler, S. B.: Use of parenteral fluids. Surgery, 1:964-976, (June) 1937.
37. Orr, T. G.: Use and abuse of intravenous therapy in surgery. Jour. Missouri Med. Assn., 34:219-222, (July) 1937.
38. Orr, T. G., and Haden, R. L.: Chemical changes in the blood of the dog in experimental peritonitis. Jour. Exper. Med., 48:339-342, (Sept. 1) 1928.
39. Rademaker, Lee: The cause and elimination of reactions after intravenous infusions. Ann. Surg., 92:195-201, (Aug.) 1930.
40. Ravenel, S. F.: The hazards of intraperitoneal injections. Jour. A.M.A., 100:473-475, (Feb. 18) 1933.
41. Rumold, M. J.: Experimental pulmonary embolism associated with venoclysis. Arch. Surg., 30:685-701, (Apr.) 1935.
42. Seibert, F. B.: Fever-producing substance found in some distilled waters. Am. Jour. Physiol., 67:90-104, (Dec.) 1923.
43. Sollmann, Torald: A Manual of Pharmacology. Ed. 3, Philadelphia: W. B. Saunders Company, 1926, p. 842.
44. Stewart, J. D.: Fluid therapy in surgery: a critical review. New England J. Med., 215:53-59, (July 9) 1936.
45. Strauss, Ludwig: Thrombose und Lungenembolie nach intravenöser Dauertropfinfusion. Zentralbl. f. Chir., 59: 909-913, (Apr. 2) 1932.
46. Studdiford, W. E.: Severe and fatal reactions following the use of gum acacia glucose infusions. Surg., Gynec. and Obst., 64:772-784, (Apr.) 1937.
47. Thompson, S. A.: Preparation of dextrose and saline solutions and apparatus for intravenous and subcutaneous use. Am. Jour. Surg., 22:127-134, (Oct.) 1933.
48. Van Slyke, D. D.: Acidosis and alkalosis. Bull. New York Acad. Med., 10:103-137, (Mar.) 1934.
49. Woodyatt, R. T., Sansum, W. D., and Wilder, R. M.: Prolonged and accurately timed intravenous injections of sugar: a preliminary report. Jour. A.M.A., 65:2067-2070, (Dec. 11) 1915.
50. Zunz, E., and Govaerts, P.: Recherches expérimentales sur les effets de la transfusion dans les divers états de collapsus circulatoire. Bull. Acad. roy. de méd. de Belgique, 29:796-854, 1919.

CARBON MONOXIDE. A PUBLIC HEALTH HAZARD*

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CARBON monoxide is a tasteless, almost odorless, colorless, neutral gas with a specific gravity of 0.97. It is practically insoluble in water and has an affinity for hemoglobin of about three hundred times that of oxygen.[19]

Source.—Ordinarily, CO does not appear in nature, but results almost entirely from incomplete oxidation of carboniferous material. The common sources listed in the literature are: the exhaust gases from internal combustion engines, especially motor cars, open fires in furnaces and stoves, leaky gas connections, alcohol stoves, and gas logs.

It has been reported to have been formed in large amounts during severe electrical storms and to have been produced by growing kelp. It is present when buildings burn, is produced in lime, brick, and charcoal kilns, is present following explosions and fires in mines, and is also produced on detonation of high explosives.[19] In the laboratory, it is produced by heating formic acid or oxalic acid with sulphuric acid.[19]

It is found in smoke, and in compartments which have been painted with oil paints and sealed.[20] It has even been reported formed from burning cigarettes.[12]

Coal gas contains about 16 per cent CO; blast furnace stack gas, 28 per cent; mine air after dust explosions, 1 to 8 per cent; and the exhaust from automobile motors about 7 per cent.[20].

Five per cent of the cars tested on the highways by Van Deventer showed a dangerous concentration (0.03 per cent or more) of CO in the air the driver breathed.[10]

Incidence of CO Poisoning.—Carbon monoxide poisoning is probably much more common than most of us realize, especially that of the chronic type. Beck states that "carbon monoxide as a cause of accidental and suicidal deaths

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in the United States ranks second to automobile accidents."[2] Davis says that it is estimated that there are 50,000 asphyxial deaths in the United States annually and that one-half of these are probably due to CO.[9]

From 1925 to 1935, the various gas companies in the metropolitan area of New York City made 21,143 calls for CO poisoning. All of the patients were unconscious when discovered. Of these, 6,571 deaths resulted, and the remainder were resuscitated.[15]

Tests have shown the blood of an airplane pilot to be 20 per cent saturated with CO.[19]

Symptoms.—Since recognition of carbon monoxide poisoning is practically essential to preventive measures, at least to the realization of the need for them, and since the symptoms are so many and varied, they shall be discussed quite fully. They vary greatly depending on the type of poisoning.

McConnell and Spiller describe three types:

1. Acute—in which death results in a short time or the patient passes into pneumonia, or such conditions as parkinsonism, et cetera.
2. Chronic—causing headache, vertigo, memory defects, and other clinical symptoms.
3. Relapsing—due to injury of certain parts of the body (especially the central nervous system) and leading to symptoms at a later date.[8]

It is generally agreed that all symptoms result from anoxemia. Thus the variation in symptoms in different individuals is probably due to the condition of the circulatory system in the various regions of the body.

According to Sayers and others, the symptoms depend largely on the percentage concentration in the blood in relation to the duration of exposure and the accompanying muscular activity. The concentration resulting from long exposure brings about more severe symptoms than the same concentration resulting from a short exposure. Table I of acute symptoms is compiled from various reports.[9,14]

For men at rest in the presence of .03 per cent carbon monoxide in the air breathed for two hours results in 25 per cent saturation of the blood; if breathed for four hours in about 32 per cent saturation of the blood.[10]

Haldane says 0.01 per cent is the highest concentration one should have for eight hours or more. Tests on eight men showed that simple tests of reaction times, binocular vision, coördination of hand and eye were unaffected until the blood was 30 per cent or more saturated. Subjectively the men felt normal with the above saturation.[10]

TABLE I.

Percentage saturation of blood	SYMPTOMS
0–10	No symptoms except some shortness of breath on vigorous muscular exertion.
10–20	No symptoms in some cases, tightness across forehead, possibly slight headache, dilatation of cutaneous blood vessels.
20–30	Headache (usually throbbing), irritability, easy fatigability, disturbed judgment.
30–40	Severe headache, weakness, dizziness, dimness of vision, nausea, vomiting, possibly collapse.
40–50	As above, confusion and collapse more likely.
50–60	Syncope, increased respiration and pulse, coma with intermittent convulsions, Cheyne-Stokes respiration.
60–70	As above, depressed heart action and respiration, death if exposure too long.
70–80	Weak pulse and slowed respiration, respiratory failure and death.
Over 80	Rapidly fatal.

A concentration of 0.06 per cent in air may produce headache within one hour and unconsciousness in two hours, while 0.1 per cent may produce unconsciousness in a little more than one hour and may prove fatal in four hours.[20]

A concentration of 0.02 per cent CO in air, breathed for 3½ hours causes 20 per cent saturation of CO in the blood, leading to tightness across the forehead and headache.[19]

During exposure there is a prolonged period of forceful breathing leading to alkalosis by reducing the carbon dioxide of the blood.[9]

The symptoms of ordinary poisoning in the order of occurrence are: weakness, headache, nervousness, fainting, respiratory failure, and, finally, circulatory failure.[9]

White of The Mayo Clinic in a rather comprehensive report lists "tightness across the forehead, headache, malaise, nausea, vomiting, palpitation, muscular weakness, convulsions, coma and death in the order given." He also mentions myocardial weakness, cardiac infarction, and ocular changes such as disturbances of vision and amblyopia.[19]

Many other symptoms are reported. Stearns, Drinker, and Shaughnessy report twenty-two patients who exhibited electrocardiographic changes, eighteen of whom showed paroxysmal auricular fibrillation, one transitory intraventric-

ular block, two ventricular premature contractions, and one auricular premature contractions. They concluded that CO asphyxia may produce no important electrical changes and that changes in T-waves and in S-T segments occur most frequently.[17]

Beck and Suter say that CO affects principally the vascular system, resulting in dilatation of the peripheral vessels, slowing of the blood stream, and increased permeability of vessel walls with hemorrhages, perivascular infiltration, and edema. Fatal cases of epistaxis and hemoptysis have occurred; also cases of bleeding from the stomach, intestines, and kidneys. Due to vascular changes from impaired nutrition, arterial spasm followed by thrombosis and gangrene may result. The heart may even rupture. They base their study on 136 cases of chronic poisoning seen during a period of fifteen years. The chief complaints of many of these patients were referred to the cardiovascular system, the most outstanding symptoms being palpitation, dyspnea, irregularity of heart action and precordial distress. There were cases of encephalitis with the parkinsonian syndrome and cases of cerebral thrombosis. Electrocardial abnormalities were encountered that disappeared after recovery.[3]

It has been thought that CO as a causative agent of myocardial disease may help to explain the seasonal incidence of coronary thrombosis. Wood and Hedley reported ninety-four cases in autumn and winter and only fourteen in spring and summer in Philadelphia in 1935.

Beck reports a study of ninety-seven patients who were repeatedly subjected to sublethal doses of CO at varying intervals over prolonged periods. Of this group the following symptoms were recorded: headache fifty-eight, vertigo and headache forty-six, general weakness fifty-two, weakness and tremulousness in legs with ataxia thirty. Nervous and mental symptoms were present in almost all, the leading ones being feelings of depression, restlessness, anxiety, and fears, but also prominent were introspection, emotional upheavals, mental retardation with memory defects and at times confusion. Drowsiness and insomnia were frequent, paresthesia was present in thirty-six, speech defects in seven, and paraplegia in three. Vasomotor instability was common and was manifested in morbid flushing, local sweating, cold extremities, and purplish congestion of the hands and feet. Neuromuscular manifestations were common, the outstanding one being pain. Pain was found to be of the dull aching type and present mainly in the back, shoulders, epigastrium, lower abdomen and chest, or of the acutely spasmodic type causing cardiospasm in six, pylorospasm in one, uterospasm in three, anal spasm in three, ureteral spasm in two, dysuria in ten, muscle spasm (usually in the legs) in seventeen, painful spasmodic contraction in one or more of the toes in fourteen. There was fibrillation of bundles of muscles in twenty-six. The majority of the patients had digestive disturbances, nausea and vomiting being present in sixteen, and gastric analysis being deemed necessary in fifty-nine. Two patients had symptoms of gastric ulcer for which they were getting treatment, but three or four days after admission to the hospital realized spontaneous relief. Of the cardio-respiratory manifestations, dyspnea was noted in thirty-six, palpitation in twenty-seven, coughing in fifteen, yawning in five, precordial distress in fourteen (two had typical angina pectoris), bradycardia in thirty, and hypotension in forty-nine. Of the genito-urinary symptoms, nocturia and dysuria were quite common and several had incontinence. Glycosuria was present in thirteen and albuminuria in twelve. Some of the women complained of menstrual disturbances and some of the men of diminished libido. The red blood cell count was found to be over 5,000,000 in forty-four.[2]

Nichols and Keller describe a case of delirium which came on following three days of unconsciousness and one day of clearness after poisoning from a gas heater. Neurologically the patient showed cog-wheel resistance in all extremities, right-sided pyramidal tract signs, and excruciating tenderness of all the peripheral nerve trunks. Then aphasic disturbances developed; the patient could use a comb and brush but could not name them. She later learned the names as one picks up a foreign language, and learned to write script but could not print.[12] They also describe a patient in whom grand and petit mal developed a year after a suicidal attempt with gas. In the meantime he had to be kept in an institution and his intelligence quotient, which previously had been 105 on the Stanford Binet test, dropped to a five-year level.[8]

Killick describes a study in which it was

found that successive exposures to the gas not only lessened symptoms but degree of saturation of the blood changed in relation to the concentration of the CO breathed. In other words, a state of acclimatization resulted. However, no increase was noted in the red blood cell or hemoglobin count.[11]

Beck and Suter found that the tolerance of laboratory animals was increased on repeated exposures, but here an increase in the red blood cell and hemoglobin counts were noted.[3]

Dancey and Reed describe a patient who one week after exposure developed the symptoms of hebephrenic schizophrenia. Eight months later she had recovered sufficiently to go home. They describe another who one month after exposure developed a condition resembling general paresis. After four months, this latter patient had fairly well recovered except for amnesia.[8]

Cohen describes a case of speech perseveration and astasia-abasia four weeks after a coma of seventy-two hours from CO intoxication. Recovery was fairly good after three and one-half months except for some disorientation and memory disturbance, and on fatigue occasional repetition of sentences and ideas.[7]

Carbon monoxide poisoning may cause encephalitis.[1]

Ormsby describes multiple gangrene of the hands and feet following poisoning with CO. The early lesions were bullæ of the type seen in a second degree burn.[13]

Much variation is seen in pulse and temperature, especially in acute poisoning. Usually the pulse is above 76 and the temperature low. Temperature has been seen between 94.6 and 102.8.[17] The blood pressure is not affected.[5]

Dancey and Reed describe a CO syndrome. There is a history of exposure, pulmonary edema, a latent period, then clinical evidence of lenticular nucleus involvement, and the common features of an organic psychosis with amnesia, confusion, and emotional instability.[8]

In the majority of fatal cases, the patient dies of acute asphyxiation, usually without regaining consciousness. In others, he becomes apparently well and three to seven days later develops secondary symptoms referable to the heart, lungs, or central nervous system, resulting in death.[3] The most frequent cause of death, if the patient survives exposure to the gas, is pneumonia.[9]

Postmortem Findings.—Those who die a few days or more after the poisoning often show bilateral necrosis of the globus pallidus.[4, 8] Selective action on the globus pallidus may be explained on anatomical grounds since this structure is supplied by long arteries that terminate in it. When death occurs from acute poisoning the most characteristic changes are bright-red blood, which is not coagulated, the pinkish- or reddish-colored viscera, reddish discoloration of the face, diffuse reddish patches on the superior surfaces of the neck, trunk, and extremities, and pink or red hypostatic areas on the dependent portions.[4]

Nichols and Keller give the pathological picture of the brain after seven weeks as follows: The pia is thickened and the vessels show connective tissue proliferation. In the cortex, there is evidence of vascular overgrowth and many shrunken, dark-staining, pyknic nerve cells with clear spaces about them. Neuronophagic cells occur, filled with fat and pigment. There is also some micro- and macroglial overgrowth. In the subcortical white matter, there is considerable destruction of myelin. There are degenerative changes in the basal ganglia, greatest in the anterior portion of the globus pallidus, which frequently shows anemic necrosis. Vacuoles are frequently found scattered throughout the brain substance.[12]

The classic anoxemic lesions in the heart are hemorrhagic necrosis and evidence of granulation and regeneration. Macroscopic hemorrhages in the apices of papillary muscles are occasionally found. Lesions are also found in the mitral valves and in the wall of the left ventricle. Coronary thrombosis has been observed as well as thrombi in small vessels.[3]

Degenerative and atrophic conditions are occasionally found in the ependymal cells of the choroid plexus, also damage to the anterior horn cells of the spinal cord may occur.[8]

Laboratory Detection of CO.—Absolute diagnosis of CO poisoning is dependent on the finding of carbon monoxide hemoglobin. Katayama's test is one of the best. It will detect as little as 10 per cent saturation of hemoglobin with CO. The test is run in this manner: 10 c.c. of water are placed in each of two test tubes. Five drops of suspected blood are added to one, and five drops of normal blood to the other. Then five drops of fresh orange-colored ammonium sul-

phide are added to each, mixed gently, and made faintly acid with acetic acid. If carbon monoxide hemoglobin is present a rose red color will develop. The control becomes a dirty greenish brown.[18]

Hoppe-Seyler's test is less sensitive and less satisfactory but a little simpler. Place three c.c. of water in a test tube, add three to five drops of blood and one drop of 5 per cent NaOH, mix gently, and let stand one hour. Blood containing carbon monoxide hemoglobin is more or less pink, while normal blood gives a greenish-brown color.[18]

Carbon monoxide hemoglobin has a characteristic spectrum, and when present in sufficient amount (30 per cent or more) is readily identified with the ordinary "pocket" spectroscope.[18]

Carbon monoxide indicators are now available on the market which indicate by direct reading the percentage of carbon monoxide present in blood or air.[20]

Use of the spectrophotometer gives an accurate quantitative measurement of CO in blood.[6]

Absolute diagnosis is made by the pyrotannic acid method or Van Slyke method.[19]

Treatment of CO Poisoning.—First remove the patient from the carbon monoxide atmosphere. In mild cases, this, plus bed rest and observation for twelve to twenty-four hours, is all that is necessary. In the comatose patient who is not breathing, or whose breathing is weak and ineffectual, start artificial respiration at once, and keep it up until the patient is breathing normally or until rigor mortis sets in. It is advisable to use the inhalator with oxygen, or, better still, with 93 per cent oxygen and 7 per cent carbon dioxide. In very severe cases 10 to 30 per cent carbon dioxide is recommended.[9]

By inhalation of oxygen, the dissociation of CO is accomplished about twice as rapidly as with ordinary air.[4] "The stability of carbon monoxide hemoglobin is due, not to the speed of its formation, but to the slowness of its dissociation."[16]

Remember the comatose patient is in shock, so during the artificial respiration it is well to rub the patient's limbs vigorously, and apply hot water bottles and blankets to the body. If necessary give a cardiac stimulant such as caffeine or sodium benzoate.[19, 9]

Most writers say that methylene blue is of little or no value, but according to Brooks of the University of California, on her experiment with rabbits, it changes carbon monoxide hemoglobin to oxyhemoglobin in the blood very rapidly. Thus it appears as if its use should be rational and recommended.[6]

Blood transfusions are of no value unless given instantly.[19]

After revival, the patient should be taken to a hospital in an ambulance (exercise is dangerous!), and an oxygen and carbon dioxide mixture given for five to ten minutes every hour for six to twelve hours. The patient should remain in bed for several days.[9]

Solution.—Beck says that the solution for the problem of CO poisoning

> "lies in the administration of proper preventive measures through well trained industrial and sanitary engineers under the direction of municipal and state boards of health. More emphasis should be placed on public health instruction with regard to the hazard, and statistics should be more available, like those obtained from traffic accidents, in order to ascertain its real significance from a public health standpoint."[3]

It would seem that the following solution would be more comprehensive and more likely to bring about results.

1. Probably most important is that more responsibility should be placed on practicing physicians in:

(a) knowing the prevalence of carbon monoxide poisoning and suspecting the condition;

(b) knowing the symptoms, not only of acute poisoning but of chronic and relapsing poisoning also;

(c) knowing how to make an absolute diagnosis of the condition, that is, by the presence of carbon monoxide in the blood, and being adept in performing such tests;

(d) knowing first aid measures and general treatment, and avoiding mistakes. The most common mistakes made by physicians in acute poisoning are:

1. Pronouncing the patient dead because no sign of life can be detected. Eight to twelve hours of artificial respiration are needed to revive some patients.

2. Putting a non-breathing patient in an ambulance. Such a patient should not be moved until normal respiration is established.

3. The use of hypo medication. Intravenous and hypo medication are probably of no use.

4. Interfering with artificial respiration to examine a patient. It is useless and dangerous.[9]

(e) doing his part to make his patients conscious of the hazards of carbon monoxide poisoning.

The above suggestions can probably best be realized by placing more emphasis on all phases of the hazard in the medical school curriculum.

2. There should be education of the public, especially those likely to come in contact with the condition, such as firemen, policemen, and others, with regard to incidence, symptoms, and first aid. Statistics should be compiled and made available to the public.

3. Methods of accurately detecting injurious amounts of the gas in air should be developed and the public educated in detecting the gas in homes, garages, motor cars, et cetera. A possible solution of this point could be that the gas companies when installing gas in a home or apartment be required to supply each patron with a piece of apparatus for detecting leaking gas.

4. There should be more experimentation to determine better methods of treatment.

5. Finally, legislation should be enacted for the elimination of the hazard in factories, public garages, mines, and wherever there are employes. Such legislation should be supplemented by regular inspection under public health jurisdiction.

Bibliography

1. Armour, Robert G.: Encephalitis, In Text Book of Medicine by Cecil. Philadelphia: W. B. Saunders Co., 1937. p. 1424.
2. Beck, Harvey G.: Slow carbon monoxide poisoning. Jour. A.M.A., 107:1020-1029, 1936.
3. Beck, H. G., and Suter, G. M.: Rôle of CO in the causation of myocardial disease. Jour. A.M.A., 110:1982-1986, 1938.
4. Bell, E. T.: A Text Book of Pathology. Philadelphia, Lea and Febiger, 1934, pp. 38-40.
5. Brewer, N. R.: Blood-pressure reponses to acute CO poisoning. Am. Jour. Physiol., 120:91-99, 1937.
6. Brooks, Matilda M.: Mechanism of methylene blue in CO poisoning. Proc. Soc. Exper. Biol. and Med., 34:48-49, 1936.
7. Cohen, Louis H.: Speech perseveration and astasia-abasia following carbon monoxide intoxication. Jour. Neurol. and Psychopath., 17:41-47, (July) 1936.
8. Dancey, Travis E., and Reed, George E.: Mental disease following carbon monoxide poisoning. Canad. Med. Assn. Jour., 35:47-49, (July) 1936.
9. Davis, Fred F.: Acute carbon monoxide asphyxia. Virginia Med. Monthly, 64:337-339, (Sept.) 1937.
10. Forbes, W. H., Dill, D. B., DeSilva, H., and Van Deventer, F. M.: The influence of moderate carbon monoxide poisoning upon the ability to drive automobiles. Jour. Indust. Hyg. and Toxicology, 19:598-603, (Dec.) 1937.
11. Killick, Esther M.: The acclimatization of the human subject to atmospheres containing low concentrations of carbon monoxide. Jour. Physiol., 87:41-55, (June 10) 1936.
12. Nichols, I. C., and Keller, M.: Apraxias and other neurological sequelæ of carbon monoxide asphyxia. Am. Jour. Psychiat., 93:42-48, (Mar.) 1937.
13. Ormsby, O. S.: Diseases of the Skin. Philadelphia: Lea and Febiger, 1937, p. 361.
14. Sayers, R. R., and Davenport, S. J.: Review of carbon monoxide poisoning. Pub. Health Bull., 195, U.S.P.H.S., Washington, D. C., 1930.
15. Shillito, Frederick H., Drinker, Cecil K., and Shaughnessy, Thomas J.: The problem of nervous and mental sequelæ in carbon monoxide poisoning. Jour. A.M.A., 106:669-674, (Feb. 29) 1936.
16. Starling, E. H.: Principles of Human Physiology. Philadelphia: Lea and Febiger, 1936, p. 918.
17. Stearns, W. H., Drinker, C. K., and Shaughnessy, T. J.: The electrocardiographic changes found in twenty-two cases of carbon monoxide poisoning. Am. Heart Jour., 15:434-447, (April) 1938.
18. Todd, J. B., and Sanford, A. H.: Clinical Diagnosis by Laboratory Methods. Philadelphia: W. B. Saunders Co., 1937, pp. 219-220.
19. White, Joel J.: Carbon monoxide and its relation to aircraft. Proc. Staff Meet., Mayo Clinic, 11:196-199, (Mar. 25) 1936.
20. Wright, W.: Carbon monoxide poisoning, in Textbook of Medicine by Cecil, Fourth Edition. Philadelphia: W. B. Saunders Co., 1937, pp. 537-539.

THE MEDICAL LIBRARY—A LABORATORY FOR THE LITERARY PHYSICIAN

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BECAUSE of the tremendous increase in the literature of medicine brought about by rapid developments in the medical and the allied sciences, the medical library is without a doubt an important factor in modern medical practice. First, it is a storehouse for the medical literature of the past. A modern medical library, therefore, provides its patrons with an access to the progress of medicine throughout the years. This, in itself, is a significant service, for it makes it possible for every physician to know what has been done before. This is of moment not only to the physician contemplating a new investigation, but also to the clinician, because it provides him with the wealth of past experience in his combat with disease.

Second (and this is the most practical reason for the existence of the medical library), is the fact that it immediately affords the reader the latest developments in medical and surgical practice. The latest advances are published first of all in the current medical journals, which are the important holdings of medical libraries. Sometimes, as might well be imagined, the expediency with which a library can provide its patrons with

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papers on modern and tried methods of therapy may be an important factor in the saving of human life.

But the use of a library is complicated. Drs. Billings, Cushing, Davis, Finlayson, Holmes, Osler, Winfield, Wylde and others have suggested that proper understanding of the use of the library is something that should be taught. Some medical schools include in their curricula studies in medical bibliography and the use of the library. Most medical men have had neither the opportunity nor the time for such study. It was thought, therefore, that it might prove useful briefly to describe some of the fundamental concepts concerning the use of medical libraries in general.

To be used most advantageously, the library might be thought of as a physician's laboratory. It is a workshop for the literary physician, and it is just as important to him as a well equipped laboratory is to the experimental bacteriologist, the hematologic laboratory is to the hematologist, or the laboratory of surgical pathology is to the hospital pathologist. And, as in the case of a laboratory, the patron should expect to find a usable physical plant as well as to receive a minimal amount of service from the library staff. That is, the library should provide its patrons with a fair number of books and periodicals, well-lighted study rooms with comfortable chairs and desks, access to the book stacks, and the necessary indexes and bibliographic tools to help them in their work. Also, the library staff should help and offer aid to the physician engaged in literary research.

To use a library intelligently, readers should have a clear understanding as to (1) what constitutes a library and (2) what they can expect in respect to service or help in answer to their many problems. Most libraries of moderate size consist of an adequate number of medical journals and a fair collection of monographs, textbooks, medical reference books and other bibliographic aids. The size and resources of libraries are dependent, to a large extent, upon the size of the communities they serve and the money that is available to them. A large medical center such as Chicago, for example, should provide its patrons with an immense variety of materials, and conversely, a small medical library, whose resources are dependent upon the local county medical society, should not be expected to have more than a minimal number of books and journals.

A library may be said to exist for the preservation and distribution of ideas by way of the written and printed word. The preservative function of libraries includes the purchase and storage of books, periodicals, and ephemeral materials. The function of distribution embodies the methods which are available to interpret the library's resources to the reader.

Since most persons are primarily interested in the practical use of the library, what then are the librarian's methods of making these resources available? First, the library provides its patrons with the public catalogue. A catalogue in its fundamental meaning is nothing more than a list of the holdings of the library. For the sake of convenience it is generally an alphabetical file of cards, but it may be bound in book form. It may be arranged alphabetically according to subjects; it may be arranged alphabetically according to the names of authors; or it may have a lexicographic arrangement, the cards being filed with author and subject entries in one alphabet. The lexicographic arrangement is preferred and is used by most libraries because it is more convenient. To supplement the catalogue of books owned, a library may have a catalogue of its periodical holdings.

To obtain a better knowledge of the books in the library, the reader should have access to the book stacks. In research or working libraries, where it is practicable, this arrangement is especially important. No matter how carefully books are catalogued, there is no substitute for the books themselves. If books are placed on the shelves in logical sequence, the reader will obtain a fair idea of the library's holdings. This knowledge may permit him to become familiar with the contents of many books which might otherwise be overlooked. Since each library has its peculiarities, it is imperative that the reader be acquainted with the way in which volumes are arranged on the shelves.

Personal instruction by the librarian should be given to each patron on the practical arrangement or scheme by which the library books are shelved. This scheme is generally called the "classification." Theoretically, classification is the outline of knowledge, but applied to books in libraries it is considered to be an outline to follow for the shelving of books. Most classifi-

cation schemes, however, have important theoretical bases. That is, a classification scheme covering the field of medicine should attempt to outline that field. There should be an introduction to the subject. This may be supplied by reference books, directories, dictionaries, encyclopedias, bibliographies, histories, and books on bibliography. Following these, logically, come the books on the basic sciences, such as biology, anatomy, physiology, biochemistry and bacteriology. The general subject, medicine, might form the body of the classification scheme. In this section might be filed the textbooks and more extended treatises on medical practice.

Next, perhaps, as suggested by the classification plan of the Boston Medical Library, are grouped the books on the different medical specialties. These should be shelved in separate sections and would include, for example, books on pathology, syphilology, cancer, diseases of the thorax, diseases of the gastro-intestinal tract, urology, orthopedics, otolaryngology, ophthalmology, surgery, obstetrics, gynecology. The remaining sections of the classification plan might be devoted to medicine and its relationship to other endeavors. In this section might be grouped books on medicine and law, medicine and industry, medicine and the state, hygiene, and medical economics..

The next provision the library makes is to place the reference books within easy access of the reader. The reference books are the numerous bibliographic aids which might contain answers to readers' problems. Included are such useful books as the medical indexes and other bibliographies of medicine; the directories; the dictionaries and encyclopedias; the almanacs and atlases; the loose-leaf sets; and the books on medical history and biography.

Most medical libraries, it is hoped, "open their houses" to readers. To what extent they will offer further services will depend somewhat upon library policy and the size of the library budget. In some medical libraries, where there is a liberal attitude, many services are offered the readers. In other libraries, however, the reader should expect to pay a reasonable fee for such services. One of the most important services offered by most libraries is accomplished by the interlibrary loan system. Although a modern medical library may be rather complete in its holdings, there are certain books or journals which may be needed occasionally, which may not be in the library. If the reader needs such books, the librarian may borrow them from some well-stocked library. To be sure, no library will lend its treasures, such as the *Fabrica* of Vesalius or even the first edition of Beaumont's *Physiology*, and very few libraries will lend books which were printed in the Eighteenth Century or earlier. If these volumes are desired, perhaps the best solution would be a trip to the library that houses them. But photostatic copies of most valuable books may be made for the reader. To copy a book photostatically is rather costly. Photo filming of books and journals on 35 mm. continuous film stock is less expensive, providing the library has a reading machine or projector with which the patron can view the finished product. The American Medical Association* will lend the journals in its library to members and to individual subscribers of its journal in the United States and Canada. The American College of Surgeons maintains a package library service for fellows of the College without charge. This service consists of supplying, on request, reprints and clippings from journals on particular subjects. The material, although it is not complete on any one subject, must be very valuable to a fellow who lacks library facilities.

Another service offered by many libraries is the preparation of medical bibliographies. Some users of a library prefer, and wisely, I believe, to look up their own material. These readers will need help, at first, from the librarian on the use of the medical indexes. Other readers feel that the librarian is better equipped to do this bibliographic work. Some libraries are able to provide this service free. Others cannot afford to do this and employ scholars who will do this work at a moderate cost to the patron.

In addition to the preparation of bibliographies, the special services of some libraries include abstracting and reviewing the literature, translating articles, and collecting case reports. If the physician finds that these services are not available at his library, he may communicate with private agencies who will do this work for him. The American College of Surgeons provides these services for its members for a modest

*The American Medical Association lends periodicals for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1928 to date. Requests should be accompanied by stamps to cover postage.

fee. Certain private medical publishers maintain bibliographic and research departments which can be used by physicians. Names and addresses of such publishers may be obtained from the American Medical Association.

To aid readers in their understanding of medical bibliography, formal lectures are sometimes offered library users. Such lectures consist of comments on the chief bibliographic tools, the importance of complete bibliographic description of references to the literature, and a few rules on bibliographic citation.

Another important service a library can offer is the recording of references to the literature as they appear. Because of the large number of periodicals indexed and the mechanical problems of indexing, it is impossible for bibliographers and publishers to keep the indexes up-to-date. A few libraries keep an index of the articles of the leading medical journals as they are published. Some librarians record new articles on particular phases of medicine at the request of the library user.

The librarian can render other services, such as the location of addresses of physicians in Europe, biographic sketches, bibliographic information about new and old medical books, in addition to the ordinary matters of library economy. Mention should be made of questions of nonmedical character. If the library is equipped for such services, the library user may be able to find answers to some of his questions. However, such questions can be answered better by the public library.

References

1. Billings, J. S.: Our medical literature. Tr. Internat. M. Cong., 7 (pt. 1):54-71, 1881.
2. Cushing, Harvey: The doctor and his books. Am. Jour. Surg., 4:100-110, (Jan.) 1928.
3. Davis, Lincoln: The value of the medical library in medical education. New England Jour. Med., 213:5-7, (July 4) 1935.
4. Finlayson, James: Library demonstrations in the teaching of the history of medicine. Janus, 8:190-192, 1903.
5. Holmes, Bayard: The medical library and its influence on medical culture and remuneration. Am. Med., 16:480-483, (Sept.) 1910.
6. Osler, William: Remarks on the medical library in postgraduate work. Brit. Med. Jour., 2:925-928, (Oct. 2) 1909.
7. Winfield, J. M.: The medical library as a factor in medical education. M. Library & Historical Jour., 2:183-187, (July) 1904.
8. Wylde, C. F.: Practical education of the medical student in library methods. Bull. Med. Library Assn., 15: 27-30, (Oct.) 1925.

TRAUMATIC INGUINAL HERNIA

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PROTRUSION of some portion of the abdominal contents through a weak spot or cleft in the abdominal wall is, and always has been, one of the most common surgical ailments. The terms rupture and breach are survivals of the old idea that the protrusion was actually due to a tear through the peritoneum. It has only recently been realized that although increased intra-abdominal tension, due to lifting, straining or active exercise generally, is often the immediate exciting cause of the protrusion, a hernia would rarely occur were there not present a preformed sac or at least an abnormal relaxation, in an existing cleft or opening. This is particularly true of the inguinal canal, as embryology has shown. And since herniations in this region have incapacitated active men from time immemorial, they have always excited the greatest surgical interest.

McGavin states that the opinion is becoming more general that many cases hitherto regarded as acquired, are in reality congenital.

Sir Arthur Keith contests the theory of preformed or congenital sacs in the origin of herniæ. He holds that herniæ which occur in adult men and women are all of them, or almost all of them, of gradual production. Repetitions day by day of straining at stool, stooping, lifting, coughing and other bodily movements cause the abdominal contents to beat against their containing walls and gradually to evaginate the weakest points of the abdominal wall, thus in the course of time giving rise to hernial protrusions. Then, he states, a sudden effort which in a normal man would be harmless, turns a partial or incipient hernia into a real one.

Ochsner notes that there can be no doubt about the existence of an hereditary tendency to hernia in many families. He affirms, however, that the chief exciting cause is increased or abnormal

intra-abdominal pressure which may be very violent and of only short duration as in lifting heavy weights, falling a great distance or with great force, violent coughing or sneezing. In such cases the tissues are virtually torn, making an opening through which the hernial contents protrude.

De Quervain states, "Most so-called traumatic herniæ are of the indirect inguinal variety, but all forms of hernia have at times been ascribed to injury. We may, however, discard the possibility in direct inguinal and umbilical hernia and should be very skeptical with reference to femoral hernia."

The hernia problem is one of perennial interest and difficulty to industrial boards. A vast number of claims are based on this abdominal lesion, many of them no doubt without any sound compensable basis, and some certainly fraudulent. A considerable body of surgical opinion holds that traumatic hernia is of rare occurrence and must be associated with laceration or actual wounding of the abdominal wall and that all herniæ outside of this small class must be considered as congenital in origin.

The attitude taken on the part of the industrial courts, however, is that trauma, while perhaps not always responsible for the occurrence of the hernia, at least acts in the capacity of a contributory force or as it is commonly phrased, an aggravation of a pre-existing condition.

Inguinal herniæ occur in the weakest portion of the anterior abdominal wall and are the penalty which the human male pays for assuming the upright position. The inguinal canal normally is a more or less obliterated passage through which the testicles migrated from their original position within the abdomen to their postnatal position in the scrotum. In the progress of its descent, the testicle was preceded by a layer of peritoneum which was pushed down before it. Ideally, this tract is completely obliterated but it is repeatedly found at operation or at autopsy in individuals who have no recognizable hernia that remnants of the peritoneum which accompany the descent of the testicle still remain in the inguinal canal constituting what is commonly called a preformed sac. This sac can in many instances be demonstrated by X-ray in the presence of a pneumoperitoneum.

Some writers go so far as to conclude that traumatic hernia does not exist. They base their opinion upon the observation that few, if any, herniæ follow direct violence to the abdominal wall such as crushing injuries, blows of one type or another with blunt objects, etc. Others base their conclusions of the impossibility of traumatic hernia on the statistics compiled on a large series of laborers passing through routine examination with follow-up thereafter in which they found that those men who subsequently developed herniæ had relaxed external rings or cough impulse, or both, on their initial examination.

On what basis are we then to determine whether or not a given hernia may be considered to be traumatic in origin? Moorhead, Kaufman and others have laid considerable stress upon the fulfillment of certain criteria before any hernia may be considered traumatic: (1) Adequate violence. This presupposes intra-abdominal pressure of an extreme degree; not the repetitions of ordinary living but the result of some very unusual act. (2) Adequate effects. These should be immediate. Pain is the usual symptom and is often of the testicular variety sufficient to cause immediate syncope, nausea and sometimes vomiting. Swelling results promptly and often this is accompanied by edema and ecchymosis. Tenderness on pressure should be present. (3) Operative findings. Some extraordinary finding such as ecchymosis, tearing of the peritoneum, edema or other evidence of recent injury must be present.

Those herniæ following strain, heavy lifting, violent coughing, etc., can hardly be classed as being due to trauma. However, if there are present the criteria of adequate violence with adequate effects as listed above, together with surgical findings consistent with the history of the accident, we feel a diagnosis of traumatic hernia is perfectly justified.

Case Report

J. N. H., a man twenty-five years of age, was injured shortly before admission to the hospital in a fall from the top of a grain elevator, a distance of about sixty feet, in which he landed in an upright position. He was rendered unconscious and was found a short time later by fellow workers. He was suffering excruciating pain and was transported to the nearest doctor's office where examination revealed the presence of an irreducible mass in the right inguinal region, a comminuted compound fracture of the right os calcis and a compression fracture of the first lumbar vertebra.

The patient stated that to his knowledge he had never had a hernia preceding the accident. The ring on the opposite side was not relaxed and there was no cough impulse.

He was transported to the Hospital as soon as he had sufficiently recovered from shock where an emergency herniorrhaphy was performed. At operation it was found that the mass which protruded from the internal ring consisted of a large quantity of edematous omentum with the peritoneal covering thereto torn in several places allowing the abdominal contents to lie outside of the peritoneal cavity. Attempts at reduction were unsuccessful until the internal ring had been enlarged. There was no evidence of ecchymosis.

Conclusions

1. Traumatic inguinal herniæ are of exceedingly rare occurrence.

2. The ordinary industrial hernia should not be considered traumatic.

3. Only those herniæ in which there has been adequate violence together with adequate effects and operative findings consistent with the history of the accident should be placed under this classification.

4. We feel that the case which we are herewith reporting falls in this group.

Bibliography

1. Callender, C. L.: The inguino-abdominal region. Surgical anatomy. Philadelphia: W. B. Saunders Co., p. 401, 1934.
2. Homans, John: Hernia and the abdominal wall. Textbook of Surgery. Baltimore: Charles C. Thomas, p. 988, 1931.
3. Moorhead, J. J.: Traumatic inguinal hernia. Jour. Am. Med. Assn., 98:1785, (May 21) 1932.
4. Moorhead, J. J.: Relation of trauma to hernia. New England Jour. Med., 209:568, (Sept.) 1933.
5. Penhollow, D. P.: An unusual case of traumatic hernia. New England Jour. Med., 211:492, (Sept.) 1934.

PSEUDO-DIAPHRAGMATIC SHADOW DUE TO PLEURAL FLUID*

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IN A PLEURAL cavity free of adhesions, fluid will first accumulate in its posteroinferior and lateral portion when the patient assumes an upright position. As the amount of fluid increases, it ascends in the pleural cavity so as to be seen best in the lateral periphery of the chest, as a ribbon-like shadow in the axillary line. The fluid also extends medially so as to obliterate various portions of the diaphragm, depending on the extent of the effusion. It has been assumed that the hydrostatic pressure of the fluid, the retractile force of the lung, the capillarity of the pleura and the negative intrapleural pressure account for this usual distribution of fluid. Occasionally, however, free fluid, instead of distributing itself in this manner, will gravitate to the inferior-most portion of the pleural cavity and insert itself between the inferior surface of the lung and the diaphragm. Thus, the superior surface of the fluid in contrast with the aerated lung gives the impression of an elevated diaphragm.

Rigler[3] has shown that transudates and most pleural exudates move considerably with change in position, and that pleural exudates both purulent type and non-purulent which have been present sufficiently long to produce adhesions may not change in their distribution even with extreme changes in position. His work was prompted in answer to the wide differences of opinion which were to be found in the literature as to whether or not pleural effusions shift in position with change in position of the patient, as well as in answer to the questions of the degree of shift and the rapidity of the reaction. In carrying out his problem, Rigler used, in addition to the usual erect, supine and lateral views of the chest, a position which he termed the lateral decubitus position. This view is accomplished by having the patient lie on the involved side with the film in front and the x-ray tube behind. The patient is built up so that the cassette may be permitted to come sufficiently low to permit the axillary portion of the thoracic cage to be included on the film. This view is especially effective in the visualization of small quantities of fluid. It can also be used to indicate the mobility and the rapidity of movement of fluids, as well as to determine whether certain shadows are due to pleural effusions which have distributed themselves in an unorthodox manner.

Atypical distribution of pleural fluid has been called to our attention before and is not looked upon as anything new. Westermark[4] in 1935

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added thirteen cases to a group of five cases reported by Jacobeus and himself in which free pleural fluid had arranged itself, as we described above, in what he termed a paradoxical manner. He called attention to the fact that others noticed that fluid had behaved in this same manner but had not been able to explain this phenomenon. Free fluid usually arranges itself in the pleural cavity so that its upper border extends obliquely medialward and downwards. The usual situation of the free pleural exudate is determined by the maintenance of those normal factors governing the distribution of fluid. In all of Westermark's cases there was an obstructive atelectasis due most often to cancer of the lung; others were due to tuberculosis, pneumonia, bronchiectasis, and trauma. In atelectasis there is an increase in the negative pressure in the pleural cavity. Westermark thought that the alteration in pressure relations in obstructive atelectasis influenced the situation of the pleural exudate, and accounted for the paradoxical situation of the fluid. The location of the atelectasis also governed the distribution. Korol and Scott[1] attempted to account for this atypical distribution on the basis of concealed air-pockets which they were able to bring to light only in the lateral decubitus position with the involved fluid-containing side uppermost on the plate.

The explanations of Korol and Scott and that of Westermark are basically the same, in that they both depend on altered elasticity of the lungs for the resulting changes in the distribution of fluid. Korol and Scott speak of hidden air-pockets, which are actually cases of hydropneumothorax. In pneumothorax, the usually negative intrapleural pressure becomes positive. When this happens the lung loses its elasticity and collapses, the capillarity of the pleura being lost. The fluid which is present is influenced only by gravity and accumulates in the inferiormost portion of the pleural cavity, very much as fluid in a barrel. In Westermark's cases, the obstruction to the bronchus, whether intrinsic or extrinsic, caused an atelectasis with a resulting increase in the negative intrapleural pressure. It would appear then that the atelectasis, which is the common factor in both situations, permits the fluid to gather as it does.

The phenomenon of pseudo-diaphragmatic shadows was first noted by the author in a case of bilateral serous exudate of unexplained origin. The fluid on the right side had arranged itself in pseudo-diaphragmatic fashion whereas the fluid on the left side, which was much less in amount, behaved in the usual manner. This has also been noted in cases of carcinomatosis, heart failure and in one instance of lymphoblastoma with marked mediastinal glandular enlargement. In none of these cases was there any evidence of atelectasis or pneumothorax except in the case of lymphoblastoma where the enlarged mediastinal nodes may have caused a partial atelectasis of the right lung. In our experience this atypical distribution of pleural fluid has been seen most often in heart disease where repeated decompensations producing fibrosis may be the factor in altered elasticity of the lung. The explanation of altered elasticity in the other types of cases is not apparent to us unless the disease process which causes the fluid also affects the elasticity of the lung.

Roentgenograms of the chest where fluid is atypically distributed give the impression of an elevated diaphragm. On fluoroscopy, with the patient in the upright position, the diaphragm will also appear to be elevated. On inspiration, movement of the pseudo-diaphragm is often quite normal or very slightly restricted, but not lost. The lung on this side of the chest will usually be as radiable as the opposite normal side. There is no succussion splash as in hydro-pneumothorax when the patient is shaken. No other findings are to be noted when these patients are fluoroscoped in the upright view. In all our cases, the fluid had formed in the right pleural cavity. Yater and Rodis[8] reported a case in which the fluid had gathered in a similar manner in the left pleural cavity. On screening in the upright position, they observed a fluid wave due to the transmitted heart impulse. On fluoroscopy with the patient in the supine position, there was always a diminution in translucency of the hemithorax on the fluid-containing side due to the fact that the fluid spread itself over the surface of the lung from base to apex. The supposedly high diaphragm was no longer to be seen but the true diaphragmatic shadow could sometimes be visualized. The fluid assumed its original distribution when the patient was returned to the upright position. Films made in the supine and lateral decubitus positions demonstrated that the fluid behaved as all free fluids were shown to behave by Rigler.[3] In the supine position, the fluid

spread from apex to base, whereas in the lateral decubitus position the fluid arranged itself along the lateral periphery of the chest between the pleura and the lung. The case of lymphoblastoma with effusion is interesting enough to be reported in detail.

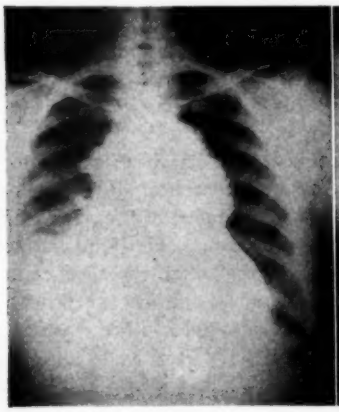

Fig. 1.

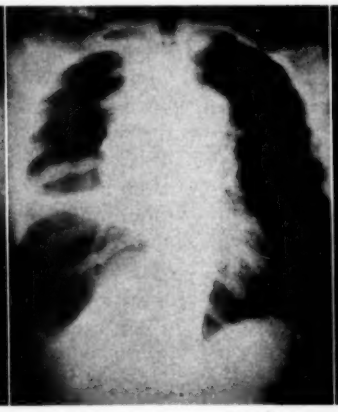

Fig. 2.

Fig. 3.

Fig. 1. Roentgenogram of chest, postero-anterior view, upright position. The mediastinal shadow is markedly widened from the enlargement of the lymph nodes. Note the shadow at the base of the right lung simulating exactly a high right diaphragm.

Fig. 2. Roentgenogram of chest, supine position. The shadow at the base of the right lung has changed radically from its appearance in the upright position (Fig. 1). It is obvious that fluid was present over the right diaphragm which in the supine position has spread diffusely over the lung. Note particularly the extension of fluid into the mediastinal pleural space and into the interlobar fissures, producing a bizarre appearance with a triangular shadow at the base

Fig. 3. Roentgenogram, postero-anterior view, in the right lateral decubitus position reveals that the fluid has accumulated along the lateral chest wall and has displaced the lung so as to produce a dense shadow between them.

Case Report

The case is that of a white woman, aged 54, who first complained of pain in the chest to the right side of the sternum nine months previous to admission. The pain was of a dull, aching character and of an intermittent type. There was a period of about three months of complete freedom from pain. One month prevous to admission, the patient developed a hacking cough which was present mostly at night. The patient raises little or no sputum and has never raised any blood. She has been growing weaker, and has experienced dyspnea on slight exertion.

Physical examination revealed a well-nourished, well-developed white female, whose only positive findings were in the chest. There was an increase in the dullness of the superior mediastinum with evidence of fluid in the right pleural cavity. The urine, blood and blood Wassermann tests were negative.

Roentgenogram of the chest revealed a widened mediastinal shadow together with what appeared to be an elevated right diaphragm (Fig. 1). The patient's chest was then fluoroscoped because of an established routine in chest cases revealing elevated diaphragmatic shadows on film study. During this procedure in the upright view the supposed right diaphragm moved slightly. Both lung fields were equally well aerated. Fluoroscopy in the supine view revealed decreased radiability of the right lung and a vaguely visualized right diaphragmatic shadow on practically the same level as the left side and not elevated as originally suspected. When the patient was returned to the upright position, the findings were again the same as seen initially. These fluoroscopic procedures were repeated many times but with no change in the findings. It was apparent that we were dealing with fluid which accumulated between the under surface of the lung and the diaphragm.

Roentgenographic study with the patient in a supine position (Fig. 2) revealed that the fluid had spread from base to apex, producing a change in the translucency of the right chest as compared to the left. Some fluid has also been sucked into the horizontal fissure and some has spread itself along the mediastinal portion of the pleural cavity to produce a trangular shadow on the right side inferiorly. Study in the right lateral decubitus position (Fig 3) revealed that the fluid had accumulated between the lateral wall of the thorax and the lateral surface of the lung.

On thoracentesis 1,000 c.c. of blood-tinged fluid was removed. Some of this fluid was centrifuged and blocked. The pathologist reported, "There are scarcely any cells, but those present seem to be markedly lymphocytes. There is nothing suggesting tumor tissue." Conclusion: "Mild chronic inflammatory exudate."

It is suggested that all patients in whom studies of the chest reveal what appears to be an elevated diaphragm be further examined in order to determine the possibility of a pleural effusion in which the fluid has arranged itself between the inferior surface of the lung and the dome of the diaphragm. This is especially im-

portant in cases in which the elasticity of the lung has been altered so as to produce atelectasis more readily, as in cardiac conditions, in instances of atelectasis by intrinsic or extrinsic pressure on a bronchus as in carcinoma or mediastinal tumors, or in cases of pneumothorax where the air has become concealed.

Conclusions

1. Fluid in the pleural cavity usually accumulates in its most dependent portion with an upper level which extends obliquely medialwards and downwards.

2. The usual distribution of fluid is due to hydrostatic pressure, capillarity of the pleura, elasticity of the lung and the negative intrapleural pressure.

3. A change in the elasticity of the lung will cause fluid to arrange itself atypically so as to simulate a high diaphragm.

4. Fluoroscopic or film study with the patient in a supine position as an adjunct to the usual upright film will bring this condition to light.

Bibliography

1. Korol, E., and Scott, H. A.: Veiled air bubble in hydropneumothorax. Am. Jour. Roentgenol. and Radium Therap., 33:777, 1935.
2. Rigler, L. G.: Roentgen observations of the movement of pleural effusions. Am. Jour. Roentgenol. and Radium Therap., 25:220, (Feb.) 1931.
3. Rigler, L. G.: Atypical distibution of pleural effusions. Am. Jour. Roentgenol. and Radium Therap. 26:543-550, (May) 1936.
4. Westermark, N.: Situation of pleural exudate in obstructive atelectasis of lung. Acta Radiologica, 16:345-352, 1935.
5. Yater, W. M., and Rodis, I.: Unusual case of pleural effusion simulating elevation of diaphragm. Am. Jour. of Roentgenol. and Radium Therap., 29:813, 1933.

CASE REPORT

SUPPURATIVE PANCREATITIS*

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IT is not my intention to review the subject of pancreatitis, because it is so well covered in recent literature, but, rather to confine myself to a report of a case of acute suppurative pancreatitis. I wish to report this case for two reasons, first, because the mortality without operation is so high, and, second, because this case exhibits some factors not encountered in other cases.

O. S., male, aged fifty-one, was first seen April 7, 1935. He complained of a pain in the upper right quadrant which had come on the day previous.

Cholecystectomy had been performed in 1930 for empyema of the gallbladder and innumerable small stones were present.

The next day he was slightly jaundiced, and the urine was dark. Three days later he felt better, there was less jaundice and the urine was lighter. Three days after that he suddenly developed a severe upper abdominal pain with nausea and vomiting. Then he was tender in the epigastrium. The next day the urine showed the presence of 3 plus bile, the scleræ were yellow, but he had less pain.

The jaundice disappeared, the urine cleared up and the stool was a brown color. A week later on April 25, he had again a severe colic at 6 a.m., and became jaundiced. The pain attacks and jaundice became increasingly worse.

On April 28 the patient was removed to Midway Hospital. Temperature and pulse were normal. I felt that he had a stone in the common duct which acted as a ball valve and operated on him April 30, 1935, under ether ethylene anesthesia. Incision was made through the old incision which had a postoperative hernia. The liver appeared swollen. The head of the pancreas was hard and this hardness extended onto the body. The head of the pancreas also was exceedingly large so that it lay over the area where one would search for the common duct. Adding to our difficulties, the first portion of the duodenum was firmly fixed high under the liver. By means of careful and tedious dissection we finally managed to free the duodenum, which had an acute angulation but no ulcer of perforation. All the tissues were very edematous and the overlying duodenum was quite unrecognizable. There now appeared only a small area where the common duct might be. The large hard pancreas head and the edema of all the structures were difficult barriers. By means of fine needles, I tested out the area and twice I drew arterial blood and once venous blood, but never bile. The only correction I had succeeded in making was to free this fixed duodenal loop. The head of the pancreas was as hard as cartilage, but did not fluctuate. A biopsy was now made of the pancreas. Drainage was established. The abdomen was closed with special attention to the post-operative hernia.

My operative diagnosis was carcinoma of the pancreas because of the hardness and size of the gland. Whether there was a stone in the common duct I did not know. Upon releasing the high hung duodenum there seemed to be a mechanical freedom as if a tension had been released on something. I had hoped to probe the common duct and establish a T tube drainage, but it could not be isolated. I was now hoping that the release of the duodenal tension would give us the necessary mechanical help and free the bile. However, if

*Presented before the Saint Paul Surgical Society, November 10, 1938.

the biopsy proved to be malignant, the outlook would be unfavorable. The post-operative course was better than expectations. In five days the jaundice had nearly all cleared up, the abdomen was soft and the stool was brown. The temperature was normal and the patient felt well.

On the eighth day, the temperature rose to 101, and the patient again became jaundiced. On the thirteenth day there was no jaundice, the stool was brown, the abdomen soft.

The biopsy report by Dr. E. T. Bell was suppurative pancreatitis, and he dismissed me with these words, "If that patient lives a year the case should be reported." It is now four years.

On the thirtieth day he went home. His appetite and sleep improved. He rapidly regained his health and strength and has worked as a department manager ever since. There have been no attacks of pain or jaundice since.

In the following brief discussion I will refer frequently to references made by Golder McWhorter in his exhaustive article.*

McWhorter divides pancreatitis into three main divisions:

A. Acute idiopathic pancreatitis:
   1. Simple edematous or nonhemorrhagic pancreatitis.
   2. Hemorrhagic pancreatitis.
   3. Necrotic or gangrenous pancreatitis.
   4. Suppurative pancreatitis.

B. Acute pancreatitis with malignancy.

C. Acute pancreatitis following trauma.

Trypsinogen as secreted by the pancreas has no digestive power and is tolerated by the pancreas.

Senn and Flexner report that if the pancreatic ducts are cut and the inactivated juices allowed to escape into the peritoneal cavity there will be no necrosis and no clinical symptoms.

Trypsinnogen becomes activated in the presence of bile in the duodenum into trypsin.

Trypsinogen also becomes activated in the presence of infection. If infection is present within the pancreas an activation of trypsinogen into trypsin takes place which would be followed by hemorrhage or necrosis. In the same way, if a small amount of bile escapes into the pancreas due to some obstruction of the ampulla of Vater, a necrosis of the blood vessels and acini would follow.

Schönbauer reports that pancreatic juice may back up into the biliary tract and gallbladder and here the trypsinogen become activated into trypsin and thus causes a necrosis and gangrene of the gallbladder. Trypsin has been found in the gangrenous gallbladder. I recently operated on a patient with acute cholecystitis and cholelithiasis, who had gangrene of the cystic duct. Small amounts of bile introduced into the pancreas would, through the activation of trypsinogen into trypsin, cause a necrosis, hemorrhage and death.

Acute pancreatitis has its onset very commonly about an hour after a full meal.

Archibald has shown that bile can enter the pancreatic ducts in two ways:

A. By obstruction of the common duct when pancreatic and bile ducts are joined.

B. By regurgitation of the duodenal contents.

When no gallstones are present, obstruction may be due to a spasm of sphincter of Oddi, a congestion, swelling, or even intestinal parasites.

Wildegans made the observation that where the blood sugar level is over 300, the outcome is fatal.

Experimentally it has been shown by Villaret and Besancon that magnesium sulphate in the jejunum produces a bile without pancreatic juice while dilute hydrochloric acid produced the opposite effect.

In suppurative pancreatitis, complicated by jaundice, the surgical mortality is well over 55%.

The controversial question for or against immediate operation is staunchly supported by master surgeons on both sides.

Körte reports 103 cases of acute pancreatitis operated early with a 60 per cent mortality. He advocates early operation. Of seventy-seven patients with various types of pancreatitis not operated on, all died.

It is generally considered by all authorities that following a cholecystectomy, pancreatitis is less frequent. In our cases the gallbladder had been removed five years previous. Stones could not be found in the common duct. The interesting mechanics of a duodenum hooked up tense to the under surface of the liver causing a mechanical difficulty and jaundice, then suddenly released with immediate relief of jaundice and pain, was dramatic.

*McWhorter, Golder L.: Acute pancreatitis. Arch. Surg., 25: 958-990, 1932.

TUBERCULOSIS DEATHS AMONG YOUNG WOMEN

The excess of deaths from tuberculosis among young women over that of males of the same age has long been regarded as an enigma by the medical profession. In an exhaustive study made in New York and Detroit, every death during one year from tuberculosis among young women was carefully investigated and several facts emerged from an analysis of the material obtained.

School life, race, nativity, participation in industrial life, insufficient clothing, poor food habits including the ever-present dieting fads, lack of sleep and too much recreation seem negligible in their influence. The real hazard is the psychic and physical changes attendant upon adolescence and maturity. Early marriage and child-bearing increase the death rate from tuberculosis in this group.—NICHOLSON, E., Study of Tuber. Among Young Women, N.T.A. Social Research Series No. 7.

HISTORY OF MEDICINE IN RAMSEY COUNTY

BY J. M. ARMSTRONG, M.D.

(Continued from August issue)

BIOGRAPHIES

William H. Morton

Dr. William Morton was a slight, gay, young man when he arrived in Saint Paul and became very popular. He had means, which perhaps was unfortunte for his professional career, as it prevented his attending strictly to business. He was a good diagnostician and a clever operator, and was considered in his day the foremost surgeon in the territory and state. He practiced first with Dr. Potts, then went into the drug business as he was too lazy, so it is said, to continue active practice. Not making a success of the drug business, he returned to medicine.

Doctor Morton's youth was spent in Paterson, New Jersey, and his medical education was probably obtained in New York. It is said his father was an Englishman and lived in Charleston, North Carolina, and that his mother died when he was a few weeks old. He was somewhat below the average in height and grew quite stout. He was appointed Surgeon to the First Minnesota Infantry, February 2, 1862, and resigned June 23, 1863, to become Medical Inspector of the 2nd Corps and shortly afterward returned to Saint Paul. About this time he was a partner of Dr. Alfred Wharton. Morton died in Saint Paul March 21, 1866, being about thirty-seven years of age. Doctor Morton seems to have been a very well informed man but his liking for liquor made him unreliable and indifferent to his work.

John Bernard Phillips

Dr. John B. Phillips was born near Kennet Square, Chester County, Pennsylvania, on March 23, 1821. His ancestors on both sides were Quakers and early settlers in that state under the patronage of William Penn. He graduated from the literary and medical departments of the University of Pennsylvania in 1855, having entered the institution in 1850, but spent two of the intervening years in Europe attending lectures at Heidelberg, Paris, and Vienna. He settled at Cottage Grove, Washington County, in 1855, afterwards coming to Saint Paul, where he died April 27, 1877. In 1854, while traveling abroad, he was arrested in Bael, Switzerland, on suspicion of being the Italian patriot, Joseph Mazinni, and was thrown into a narrow, damp, filthy cell and put on a bread and water diet for ten days. He maintained he was an American physician. To test him his captors asked him about yellow fever. He stated he had never seen a case. His interrogators then were sure he was not telling the truth as "was not yellow fever an American disease?" The explanation that his home was in

Philadelphia meant nothing. Finally, after an appeal to the United States Consulate, his identity was established and he was released.

Doctor Phillips was a member of the Ramsey County Society and of the State Association at its inception. Being a man of means and literary tastes, the daily drudgery of medical practice and struggle for a clientele did not appeal to him, hence he soon drifted away from it. He commanded a varied and thorough knowledge of literature, was versed in the classics, and was a master of the German, French, and Italian languages. He is described as a charming, cultivated and intelligent man. He was an intimate friend of Bayard Taylor, who was a student at the university with him. He was at one time a member of the Pension Board. In the Ramsey County Medical Library are a number of certificates of his attendance at the clinics at the Paris hospitals in 1853 and 1854; his membership certificate in the Minnesota State Medical Society in 1871, and a number of other items of his, among which are two federal licenses to practice medicine in 1866 and 1867. He was State Commissioner of Statistics in 1876-1877.

Azariah Theodore Crane Pierson

Dr. Azariah Pierson was known as "Father" Pierson by the Masonic Fraternity. He was born near Morris Plains, New Jersey, August 29, 1817. In 1837 he graduated in medicine at the old Barclay Street Medical School in New York. He came to Saint Paul early in 1851 in the employ of the Indian Department. For a number of years previous to 1885 he served as chief draughtsman in the office of the Surveyor General of Minnesota. It is doubtful whether he ever engaged in the practice of medicine, but for some years he was in the drug business in New York. He died in Saint Paul, November 26, 1889. The latter years of his life were devoted to Free Masonry. He became one of the most prominent Masons in the country. He was Grand Prior in the Supreme Council of the thirty-third degree Masons.

Russel Post

Russel Post came to Saint Paul in 1855. Assuming the prefix "doctor" he began doctoring by concocting medicines out of roots and herbs, but soon abandoned that practice and healed the sick by "laying on of hands." He had been a sailor, then a Connecticut peddler, and later dealt in real estate in Cleveland and in Cincinnati. His health failing he came to Saint Paul and took up the healing art. In the directory of 1856-1857 his residence is given as Pearl Street (West Fifth) between Washington and Franklin (East Twelfth Street was later also called Pearl Street). His name does not appear in the directory of 1858-1859. There was no directory published again till 1863 when Russel Post is listed as an electropathic physician with his office on Eighth Street, near Robert.

"Doctor" Post did a large and profitable business and treated his patients by placing one hand on the head and gesticulating with the other, at the same time keeping up a rapid talk on the benefits derived from the magnetic influences. He also took locks of his patients' hair and for a fee would undertake to give absent treatments, reaching the individual by holding the lock of hair in his hand, the whereabouts of the patient being a matter of no consequence. He was a venerable looking old fellow with white hair and long whiskers (in 1866) and an inveterate talker, always prying into other people's business and characterized as a nuisance by the regular profession.

"Doctor" Post was very acute in his business dealings and accumulated con-

siderable property. He owned some land where Earl Street now crosses East Seventh Street which was known as "Post's Siding." Later, at the time of his death at about the age of sixty-five years, he was engaged in exploiting a mineral spring at Bald Eagle Lake and had started the erection of a Sanatorium. After his death, his daughter, Laura Post, continued his business and professed to hold communications with the spirits of the departed. She was not as successful as her father.

Thomas Reid Potts

Dr. Thomas Reid Potts was born in Philadelphia, February 10, 1810, and graduated at the University of Pennsylvania in 1831. He practiced at Nachez, Mississippi, and for ten years at Galena, Illinois, from 1841 till he came to Saint Paul in 1849. He was married at Fort Snelling in 1847 to Miss Abbe Steele, sister of Franklin Steele (who came to Minnesota in 1837) and Dr. John Steele, and was a brother-in-law of General H. H. Sibley. At the time the First Minnesota Regiment was recruited in 1861, Doctor Potts was the Medical Examiner. Josiah R. King was the first volunteer soldier to enlist for the war, so Doctor Potts examined the first volunteer soldier of the Civil War. As soon as King was pronounced fit, he was brought out and sworn into the service in the street in front of Doctor Potts' office.

During the war, Doctor Potts was contact surgeon at Fort Snelling, physician to the Sioux in 1850-1851, and medical purveyor of the District, and also was on the first Pension Board in Saint Paul. He resided for many years on Robert Street near Sixth in a small, white, one story, frame house. He was a small man of dark complexion and brown eyes with rather pronounced features. He was popular with all classes, a great story teller and somewhat of a politician, but unfortunately was not a good business man. At one time he attempted to practice in Duluth but his old political enemy, Dr. Thomas Foster, then editor of a Duluth newspaper, prevented to a large degree any success there.

Doctor Potts was elected president of the first Town Board in 1849, was city physician and health officer in 1860-1862, and again from 1866 till his death which occurred suddenly October 6, 1874. He was first president of the Minnesota Medical Society organized in 1853 and first president of the Saint Paul Academy of Medicine and Surgery established in 1860 and a founder of the Ramsey County Society.

Edward (or Edmund) G. Pugsley

Dr. E. G. Pugsley originally came from Ohio. Where his medical education was obtained does not seem to be known. He practiced at Neenah, Wisconsin, and must have moved to Stillwater in 1853 or 1854, though it is said he was there as early as 1850. He opened a drug store in Stillwater and practiced there till the war. He was Assistant Surgeon in the First Minnesota Infantry, and was later transferred to the Ninth. After the war, he practiced for several years at White Cloud, Minnesota, and in 1904 was living in Washington State.

According to a note in the Stillwater paper, Doctor Pugsley died in Tacoma, Washington, in January, 1931, at the age of eighty-three. It is also stated that he was at one time a partner of Dr. J. K. Reimer of Stillwater.

It is evident that father and son are confused by the Stillwater paper. The father was married in Saint Paul in 1852 and the son was admitted to the Ramsey County Medical Society in 1888. No doubt, it was the son who was living in Washington in 1904 and died in 1931.

William Ray

Dr. William Ray was born at Natchez, Mississippi, January 3, 1843. He graduated as Doctor of Medicine from the Missouri Medical College in 1868, though it is evident that he practiced medicine before that time as he was a surgeon in the Confederate Army. He came to Saint Paul after his graduation and remained there about three years when he moved to Delano, Minnesota. In 1889, he removed to Philipsburg, Montana, where he resided till the time of his death, March 18, 1909. At Philipsburg, he was surgeon to the Hope Mining Company and chairman of the Board of Health at the time of his death. He was very highly thought of in Montana. He was buried in Saint Paul.

Francis Rieger

Dr. Francis Rieger was born in Berlin, though part of his youth was spent in Magdeburg. He was educated in the former place where his father, Dr. Alexander Rieger, practiced medicine. Early in the fifties, he came to America and settled in Cincinnati. Three or four years later his younger brother, Paul, came from Germany and together they came to Saint Paul. Paul Rieger was a pharmacist and opened a drug store on Saint Anthony Street in the Empire Block, and later on the corner of Saint Anthony and Washington in the Winslow House. Dr. Francis Rieger was surgeon to the 8th Minnesota Regiment. After the war he located at Leavenworth, Kansas, or some place in Missouri, but returned to Saint Paul, remained there three or four years and then went to Omaha. How long he remained there is unknown, but on account of failing health he went to Denver and finally to Las Vegas, New Mexico, where he died probably about 1885. He left two children, a son and a daughter. He was born about 1831, being about thirty-one years of age when he went into the army.

Doctor Rieger was very German in his mental attitude, had a violent temper, and was impulsive and excitable. He suffered a great deal from some stomach disorder and perhaps this may have had its influence on his disposition. He had a predilection for surgery and did it well. His quick temper and lack of a sense of humor, however, often got him into trouble with his brother practitioners as he took offense when no discourtesy was intended. As an instance of surgical fees in the early days, one might mention that in 1858 he operated for a large parotid tumor. The operation was successful (the patient, a woman, lived till about thirty years ago). His charge was one hundred dollars, quite a sum for people in moderate circumstances, especially after the monetary panic. of 1857. Surgical removal of the parotid gland was considered a very formidable operation at that time and only the most skillful and daring surgeons attempted it. Paul Rieger moved from Saint Paul to California where he established the well-known firm of perfume makers.

Gustavus Rosenk

Dr. Gustavus Rosenk was a peculiar character. His office was at one time on Saint Anthony Street near Hill in the lower part of the block, a grocery store being underneath it. In the directory of 1858-1859, his office and residence is given as Third Street between St. Peter and Wabasha. It is said he came from East Prussia and had been in the government employ in a civilian capacity as district physician some place along the Rhine. He came to America in 1851 or 1852, and to Saint Paul in 1855. Many stories were told about him which prejudiced the people against him. His first wife died suddenly of cholera it was said, and later his home burned down and one or two of his children

burned with it. Later, one of his daughters died under peculiar circumstances and although an autopsy was done nothing ever came of it. These things, together with his unfortunate inability to make friends, added to his unfavorable reputation. He was appointed Senior 1st Lieutenant, Second Battery Minnesota Light Artillery, January 18, 1862.

After his return to Saint Paul, Doctor Rosenk had his office in his home on West Seventh Street between Seven Corners and Chestnut, about where Arbogast's Butcher Shop now is. Doctor Renz said when he was a boy he was afraid to go into Dr. Rosenk's garden because it was said he had a dissecting room there and kept a tame snake in his dingy little shop. Perhaps he fostered such a belief to keep small boys out. People seemed distrustful of him but he always maintained a dignified manner, carried a cane, and wore a silk hat, although often unshaven and untidy in dress. He was very poor and his practice was among the poor. He probably never obtained a medical degree, and was known as an irregular practitioner. He went to Oakland, California, about 1888 and died there.

Sylvester J. Sawyer

The place of Dr. Sylvester Sawyer's birth was probably Hookset, New Hampshire, but his place of residence from an early age was at Keeseville, Essex County, New York, where he is buried. He died in New York City, November 25, 1870, at the age of forty-two years. The Milwaukee, Wisconsin Directory for 1859 contains his name as a physician, but at the time he entered the army under contract, November 17, 1862, as Acting Assisting Surgeon, he gave his residence as Raymond, Wisconsin. He remained in the army till January 27, 1864, when his contract was annulled at his own request. His first wife died in childbirth, in about 1859. He married again at Raymond, Wisconsin, in 1862 and had several children by his second wife.

It appears that Doctor Sawyer graduated from the medical department of Columbia University in 1854, and spent some two years thereafter studying in Paris. The *New York Medical Register* for 1871 contains an obituary notice of Doctor Sawyer, but that volume is not available for reference here. Those few residents of Saint Paul, who have any recollection of him, state that he was a man of culture and good manners. He married his first wife in Saint Paul. She was a half-breed daughter of General H. H. Sibley.

(To be continued in October issue)

President's Letter

AFTER the summer let-up in medical meetings, during which time we reviewed our relationship with the University, and the State Board of Health, we turn again to the program of the month as outlined for the year. This month the program deals with the subject of accidents and their prevention.

September was selected because the annual figures show that there are more automobile accidents in September than in any other month of the year. It would be interesting to speculate on the reason why. Probably your guess is as good as any one's. The Safety Council suggests increased traffic due to vacationists returning to work, and also to a greater haste and a greater pre-occupation as fall and winter plans get under way.

The question of accident prevention is largely a lay matter, the doctors not having more than the expected percentage of accidents. Based on the fact that there is only one physician to every 1,000 lay persons, roughly speaking, only one in every 999 accidents involves a doctor.

The Minnesota Safety Council, with Col. Mattson as president, Mr. A. V. Rohweder as chairman of the Board of Directors, and Mr. C. H. Zeland as Executive Secretary, have accomplished a great deal in accident prevention, and we are indebted to them for much of the material in the packet of the month.

Mr. Rosenwald, director of Safety of the Minnesota Highway Department, has also cooperated with us in the accident problem.

The doctor becomes involved after the accident has happened. Our association has a fracture committee whose main objective for the year is improvement in the emergency care of fractures. This committee is particularly interested in the handling of the accident case before expert help arrives and gladly joins with the Safety Council in its campaign to prevent the moving of accident victims by untrained bystanders.

The committee is also interested in the even more serious problem of prevention of home accidents.

Accidents in the home result in more deaths and more serious injuries than do accidents on streets and highways and in industrial plants. It is an astonishing fact that this phase of accident prevention has received so little recognition.

Most of the deaths resulting from home accidents could be avoided and our September education campaign should lay special emphasis upon safety in the home.

GEORGE EARL, M.D., *President*
Minnesota State Medical Association.

EDITORIAL

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BUSINESS MANAGER
J. R. BRUCE

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DEXTROSE AND INSULIN IN NONDIABETIC SURGICAL CASES

THERE is much of theoretical interest and practical importance in the clinical study of the metabolism of dextrose by nondiabetic individuals who are undergoing surgical procedures. The internist frequently is confronted with the question of the interpretation of glycosuria occurring after infusion of dextrose in treatment of surgical patients. The surgeon is interested in questions of the value of insulin and of dextrose in the treatment of certain postoperative complications, such as shock, acidosis and poor healing of wounds. Answers to these questions are found in an analysis of certain known facts concerning the tolerance of the organism for dextrose and the action of insulin in the nondiabetic organism.

According to data obtained from experiments on dogs and normal men, the individual having a normal ability to store and oxidize carbohydrate can receive dextrose by the intravenous route at the rate of about 0.85 gm. per kilogram of body weight per hour without excreting abnormal amounts in the urine. Thus, a normal man weighing 70 kg. should have no glycosuria if a liter of 10 per cent solution of dextrose was infused at a uniform rate during approximately two hours. The postoperative surgical patient, however, may exhibit for a variable period a diminished ability to utilize dextrose, for at least two reasons: (1) starvation, and (2) the surgical procedure itself, including the anesthesia. Obviously, there is no easy way of estimating the extent of this diminution in tolerance in any given case. Hence, the interpretation of glycosuria after infusion of glucose may be difficult and further investigation may be necessary before previously unsuspected diabetes can be definitely excluded. This investigation should consist of determination of the blood sugar after a night's fast. In cases in which doubt still remains, a dextrose tolerance test may be necessary. The patient on whom such a test is made should have fully recovered from the operation and, for several days before the test he should have been supplied with liberal amounts of carbohydrate food. In the routine management of patients after operation, however, it is possible to avoid raising a suspicion of diabetes unnecessarily if dextrose is infused at a rate well under the normal limit of utilization; that is, if a man is of average body weight it is well to take three to four hours for the infusion of a solution containing 100 gm. of dextrose. If glycosuria occurs repeatedly with such a rate of infusion, then diabetes must be strongly suspected.

In the four or five years following the discovery of insulin, in 1922, a large number of pub-

lications were concerned with the usefulness of insulin in surgical operations on nondiabetic patients. Treatment with insulin and dextrose was heralded by some as a specific in the treatment of surgical shock and postoperative acidosis. Early work which was thought to indicate diminished amounts of insulin in the tissues of dogs that had been subjected to ether anesthesia apparently provided a sound basis for the belief that insulin might be a valuable agent in postoperative treatment. Now, however, it is known that early assays of insulin in animal tissues other than the pancreas were erroneous for technical reasons, and thus some of the rationale for this use of insulin was lost. The number of publications on the surgical uses of insulin has decreased markedly in recent years.

The basis for the use of insulin in postoperative treatment seems to be the assumption that in treatment of normal individuals, as of diabetic patients, it results in the utilization of carbohydrate at a faster rate than would be possible if insulin were not administered. However, the experimental evidence for increased rates of utilization of dextrose by normal subjects under the influence of excess insulin is not striking. Furthermore, there are no clinical or experimental data available proving that production of insulin by the nondiabetic surgical patient is seriously impaired for any appreciable period. It is true that such individuals sometimes demonstrate ketosis and diminished dextrose tolerance. These may be due in part to temporary depression of production of insulin as a result of fasting. However, in most cases they can be rapidly overcome by administration of dextrose alone. Thus, with the possible exception of occasional cases in which severe ketosis and diminution in dextrose tolerance results from prolonged starvation, and certain special instances such as the temporary "diabetes" which may occur following removal of a hyperfunctioning tumor of the islets of Langerhans, there appears to be little indication for the use of insulin in treatment of nondiabetic surgical patients. When it is used, it should be administered with good judgment and careful laboratory control in order to prevent episodes of hypoglycemia which the patient might be poorly able to stand.

RANDALL G. SPRAGUE, M.D.,
Fellow in Medicine, The Mayo Foundation, Rochester, Minn.

OVERCROWDING OF THE MEDICAL PROFESSION

There are more physicians in relation to the population in our country than in any other country in the world. While the general population has reached a point where it is increasing very slowly, the figures show that in recent years some 5,500 medical graduates are turned out each year, whereas only about 4,000 physicians die. This means an increase yearly in this ratio.

There are some who would argue that no attempt should be made to curtail the number of medical students of the country and that expansion of equipment of medical schools to care for more of the applicants should not be discouraged, the law of the survival of the fittest being allowed to apply after graduation. They perhaps do not know that each year there are some 12,000 applicants to our medical schools, only about half of whom can be accommodated. Incidentally these 12,000 applicants average about three applications apiece, and one is known to have made as high as forty-one applications, to assure admission somewhere.

Overcrowding in business leads to cut-throat competition. In a profession it leads to commercialism, something that should not be in evidence in a profession, but which is always present to some extent and is increased with financial stress. This overcrowding is detrimental to the profession and the public.

According to Bevan,* in 1904 when the Council on Medical Education was created, there were some 28,000 medical students in the country. By increasing requirements and lengthening the Medical course, the number was reduced to 1,200 by 1920. The number has been gradually increasing since then to about 23,000 in 1935.

Far be it from us to advocate lengthening the present medical course, nor of making it more difficult. In order to reduce the number of medical students, the fairer way would seem to be in being more selective in accepting candidates for matriculation in the medical course, not only from the standpoint of scholarship, but from that of character and general fitness, difficult as this latter may be to evaluate.

In discussing the question of overcrowding, the status of the proportion of Jews in the medical profession arises. This subject is at present in

*Bevan, Arthur Dean: The overcrowding of the medical profession. Jour. Am. Med. Assn., 107:337, (Nov.) 1936.

the limelight in view of the persecution of the Jews in Germany and their immigration into this country.

According to Bevan, fourteen years ago 10 per cent of students in our medical schools were Jews. In 1935 the percentage had increased to 20 per cent, and Rabbi Lazaran has found that in 1933, 32 per cent of applicants for admission to the medical schools were Jews. With 42 per cent of all Jews in the United States located in New York City, the problem in New York state is particularly difficult. The medical schools in New York City can obviously not accommodate all the Jewish students, and many apply elsewhere. That there has not been obvious discrimination against Jewish applicants to medical schools in general, is indicated by the fact that whereas only 3.5 per cent of the general population of the country is Jewish, Rypins found 17 per cent of medical students belonged to this race.

PULMONARY EMBOLECTOMY

THE operation of pulmonary embolectomy has now been performed with complete success in eleven cases, as follows:

Surgeon	*Date*	*Age*	*Sex*	*Reference*
Kirschner, M. (Konigsberg)	March 18, 1924	38	F.	Arch. f. Klin. Chir. 133:312-358 (Apr., 1924)
Meyer, A. W. (Charlottenburg)	Feb. 9, 1927	54	F.	Deutsch. Zeit. f. Chir. 205:1 (1927)
Crafoord, C. (Stockholm)	Sept. 23, 1927	54	F.	Acta. Chir. Scan. 64:172-186 (1928)
Nystrom, G. (Uppsala)	Feb. 4, 1928	35	M.	Acta. Chir. Scan. 64:110 (1928)
Meyer, A. W.	Feb. 10, 1928	n.s.	F.	Deutsch. Zeit. f. Chir. 211:353 (1928)
Crafoord, C.	Oct. 11, 1928	52	F.	Loc. cit. supra
Nystrom, G.	Jan. 23, 1929	46	F.	Ann. Surg. XCII:498-528 (1930)
Meyer, A. W.	Feb. 4, 1931	78	F.	Deutsch. Zeit. f. Chir. 231:586-592 (1931)
Crafoord, C.	Jan. 4, 1933	n.s.		Pers. comm. to. Lewis (see last ref.)
Valdoni, P. (Rome)	Nov. 27, 1935	68	M.	Policlinico (Sez. Prat.) 43:911-918 (May 18, 1936)
Lewis, Ivor (London)	Sept. 12, 1938	49	F.	Lancet, 1:1037-1041 (1939

Thus it will be seen that of the eleven operations, five (Crafoord-3, Nystrom-2) were done in Sweden, four (Meyer-3, Kirschner-1) in Germany and one each in Italy and England. The technic followed in all of them was practically that laid down by Trendelenberg in 1908, with the slight modifications suggested by A. W. Meyer after his first case.

The drawbacks to the performance of pulmonary embolectomy may be summarized as follows:

1. The diagnosis of pulmonary embolism is not always easy. Especially difficult is it, at times, to differentiate from coronary occlusion. Uremia has been mistaken for it and there are other possibilities. A wrong diagnosis has accounted for some of the unsuccessful attempts, resulting fatally.

2. Many patients with pulmonary embolism, even of severe type with most alarming symptoms, have been known to recover spontaneously and it is sometimes very difficult to evaluate these. Nystrom feels so keenly about this that he has laid down the dictum that no patient should be subjected to this operation unless death is obviously imminent. This rule has been meticulously observed in the eleven successful cases; indeed, in that of Mr. Lewis, he went so far as to wait, after opening the chest, for the patient to be in articulo mortis, before opening the pericardium and completing the operation.

3. There is one more drawback which will be mentioned very briefly, since it concerns only matters of surgical technic. The operation of Trendelenberg-Meyer presents no especial difficulty to a trained surgeon, but it entails the use of several special instruments. We believe that all of these can be dispensed with and the operation done with the instruments which are to be found in every hospital. Instead of using the special hook to pass a rubber tube through transverse sinus for the purpose of constricting the fused aorta and pulmonary artery during the incision into the latter and the removal of the clot, it would be much better, we think, to discard both the hook and the tube, passing the operator's left index and middle fingers into the transverse sinus and merely pressing the pulmonary artery with the ball of the left thumb. In this way, the most crucial point in the operation—the constriction of the aorta—is entirely avoided.

With the foundation that has been laid elsewhere we believe that it is only a question of time before we shall hear of some successful pulmonary embolectomies being done in this country, for with all the drawbacks that may be conceived there still remain some cases in which

surgical intervention is unquestionably indicated; those where the diagnosis is clear, the patient free from severe intercurrent disease and evidently in extremis, a competent surgeon at hand and enough time before death ensues to make the few necessary preparations. In these circumstances we believe that a patient in such jeopardy is entitled to his one chance and that it is the surgeon's plain duty to give it to him.—*G.C.*

MISCELLANEOUS

THE BIRTH REGISTRATION LAW AMENDED TO MEET PRESENT SOCIAL NEEDS*

AMENDMENTS of the Minnesota birth registration law, enacted into law by the 1939 Legislature, of interest to physicians, particularly to those practicing obstetrics and to those holding the official position of local registrar in cities, shorten the attending physician's time of reporting births to local registrars from ten days to five days; provide that physicians shall report births of illegitimate children direct to the State Registrar, omitting their report to the local registrar, and that the State Registrar may make and file replacement or substitute certificates under certain conditions.

The amendment providing that the physician attending at the birth of a child born out of wedlock shall report that birth direct to the State Registrar, not to the local registrar as has heretofore been required, will enable the State Registrar to coöperate more fully with physicians in the distressing circumstances which sometimes attend the delivery of the unmarried mother. The amendment will also tend to do away with the falsification of birth records of illegitimate children. The impulse to falsify these records has been motivated by a desire to protect the mother and her family without thought for the welfare of the child. But the child because of his falsified birth record will have no identifying birth certificate until his real parents, and any other person who might be falsely named in the certificate as parent, as well as the person who falsified the certificate, go before the court so that it may make a finding of facts and order the recording of those facts—an inconvenient, embarrassing, and sometimes an impossible legal procedure, if all persons concerned are not available to testify.

The amendment providing for the reporting of births of illegitimate children direct to the State Registrar contains a clause requiring the State Registrar to send an abstract of the birth report to the local registrar. It does not specify when this abstract must be sent; unless the circumstances are extenuating it will probably be sent within a month after the State Registrar receives it. The present plan is to send the local registrar a memorandum containing the surname of the child, the sex, and the date of birth, with an order to enter the data in his index only, and to refer any person making inquiry about the record to the State Registrar. With the order will go a reminder that the penalty for divulging the fact that any child is illegitimate is a crime punishable by a fine or imprisonment. Since 1915 the law has stipulated that the birth certificate of an illegitimate child is a confidential record, and that a court order must be secured before the State Registrar or any local registrar may issue a certified copy of such a record, or disclose the fact of illegitimacy. It has stipulated also that the name of the putative father must be omitted from the certificate unless he consents to its inclusion. This last provision was made so that the certificate would be left blank concerning paternity pending a judgment of paternity in the district court when the father's name would be entered in the certificate.

The purpose of sending an abstract of the births of illegitimate children to local registrars is that if the registrar receives a request for the birth certificate of a person listed in his index for which he has no certificate in his files or register, he will know definitely that a birth record was made and where it is; hence, he will be able to direct the applicant to the State Registrar for a certified copy. A second and better reason for sending the abstract to local registrars is that it will help to insure complete reporting of births of illegitimate children. Usually the registrar knows when such a birth occurs. If he ought to have an abstract concerning it, but has none, he will suspect that the birth has not been reported and will notify the State Registrar, who will take immediate steps to see that a record is made and filed. Unless the local registrar is in a position to assist the State Registrar by checking completely on his district, births of some illegitimate children will go unreported.

Should circumstances surrounding the birth of a child be particularly distressing, at the physician's request the State Registrar's report to the local registrar may be delayed. The notation of the birth of an illegitimate child in the index of the local registrar will be inconspicuous. Should the register containing the index fall into unofficial hands, the notation will not be recognized as one that concerns an illegitimate child. Furthermore, should the child become legitimized by the marriage of its parents to each other, the notation in the index will be amended, and a complete birth certificate showing the child to be legitimate will be entered in the register in its proper place.

Other amendments affecting birth records of illegitimate children are patterned after the law of Maryland, a law which has worked out well in that state; one which has been adopted by a number of other states in the country. It provides that the State Registrar may prepare and file a new birth certificate for adopted children and for those legitimized by the marriage of their parents to each other. Thus, when the court has made a decree of adoption, the foster parents may file a certified copy of the decree with the State Registrar, who will prepare a new birth certificate containing the child's new name and data pertaining to the foster par-

*Prepared by Gladys G. Casady, Assistant Director, Division of Birth and Death Records and Vital Statistics, Minnesota Department of Health.

ents. The new birth certificate will be considered the child's birth certificate from the time it is made and officially filed. A certified copy of it will be prima facie evidence of the facts contained therein. The new certificate will not disclose the fact that the child is an adopted one. These new certificates for adopted children will be in the custody of the State Registrar only. They will not be filed with local registrars or with Clerks of the District Court. They will identify the child under its legal name and as the child of its legal parents. They will be available for use for school, passport, employment, to present with claims for the payment of insurance, for pensions, and for any other purpose for which a birth certificate may be required. The original birth certificate containing the facts of birth must be preserved in the office of the State Registrar, sealed and locked in a confidential file; the seal may be broken only upon written order of the State Registrar or of the court; and not inspected by anyone other than the State Registrar, or copied only upon order of the court.

Similar to the replacement certificate for the adopted child is the replacement certificate for the child legitimized by the marriage of its parents to each other subsequent to its birth. Although the law has for a good many years declared such children legitimate, their birth certificates have continued to carry the stigma of illegitimacy. It is true that since 1923 the law has permitted the father of a child legitimized after its birth to file an affidavit with the State Registrar declaring himself to be the father of the child and the husband of the mother. The law required that the affidavit be attached to the original birth certificate, the child's surname changed to that of its father, and a notation made on the certificate that the child was legitimized on a certain data. All these notations and additions were required to be made in red ink. A certified copy, if made, contained the original facts of birth entered in black ink, lined out in red ink, and finally, the additions and changes entered in red ink, thus making the sequence of events clear to the court or to other interested persons. If the original birth certificate contained as the name of the father, a name different from that in the affidavit of paternity and legitimation filed by the man who married the mother, the affidavit could not be accepted for the purpose of amending because it did not settle the question of paternity, rather, it contradicted it. Only the court can settle a dispute concerning paternity.

Under the provision of one of the recent amendments, if a man files an affidavit alleging paternity of a child, and that paternity contradicts the paternity contained in the original birth certificate, the court may hear all evidence and testimony, make findings concerning paternity, and order both a new certificate made to contain the facts as found and the State Registrar to substitute the new certificate for the original false certificate which is in his files. Should there be no dispute of paternity, the father may file with the State Registrar proof of his marriage to the mother and his affidavit declaring his paternity. Upon receipt of these documents the State Registrar may make a new birth certificate to replace the one in which the child was registered illegitimate. The State Registrar must, however, preserve the original certificate in a confidential file, and thereafter he may not issue a certified copy of that original, or permit inspection of it unless he has a court order to do so.

The amendments discussed thus far have been for the purpose of straightening out the predicament of the adopted child and of the child who has been legitimized by the marriage of its parents to each other. Another amendment provides that the State Registrar may certify to any part or parts of an original birth certificate, and that the certified part or parts shall be prima facie evidence of the facts set forth. This means that any person born out of wedlock, not adopted and not legitimized, may have a certified copy of relevant facts taken from his birth certificate for use for insurance adjustment, for employment, for passports, or for any purpose where the fact of legitimacy or illegitimacy is immaterial.

You may ask whether or not the amendments herein discussed will help or will further handicap the child born out of wedlock. From years of ineffectual attempts under the old law to save from embarrassment individuals born out of wedlock who need their birth certificates, registration experts know the amendments of the law will ease many a difficult situation. The increasing nation-wide necessity for birth certificates to establish the rights of individuals, financial and legal, makes it imperative that a birth certificate identify the child, and that pertinent parts of it may be certified to without embarrassment to the individual whose record it is. It makes it imperative also that all original data be preserved and that a complete record of the *facts of birth* be available, should the necessity for the presentation of those facts arise.

BLOOD-STREAM INVASION BY TUBERCLE BACILLI

Blood-stream invasion by tubercle bacilli may take place at any stage of tuberculosis and at any age; following the primary infection; during the evolution of the disease or as a terminal event. The stage of the disease influences considerably the incidence of implantations in the various organs of the body; the age of the patient plays a minor rôle. Chronic forms of hemotogenous tuberculosis are uncommon in children only because, as a rule, they do not live long enough for the protracted manifestations of the disease to develop. When tubercle bacilli invade the blood-stream there may result a generalized miliary tuberculosis that overwhelms the patient in a few months or there may be a complete absence of symptoms or any variation between these two extremes. The numbers and virulence of the tubercle bacilli, the frequency of invasion, the portal of entry and the sensitivity of the body to tuberculotoxins will determine the issue.—Tubin, Eli H., Amer. Rev. of Tuber., 1939, 39.

MEDICAL ECONOMICS

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
W. F. Braasch, M.D., Chairman

WAS THE SURVEY ON THE SUPPLY OF MEDICAL CARE WORTH WHILE?

Many physicians who took part in the recent Survey of the Supply of Medical Service which was conducted by the various county medical societies of the state and nation have wondered about the results accomplished. Undoubtedly many who took the time and effort to fill out the blanks sent to them wondered whether their efforts created data which were worth while. Those who threw the blanks into the waste basket are probably satisfied that they were not. *However, the latter group were wrong.*

Inadequacies Revealed

The completion of an immense undertaking such as the National Survey proved to be is necessarily slow and laborious. The results of the Survey are still coming in from states that were delayed in making it. However, a final summary of the data accumulated will soon be forthcoming from the Bureau of Medical Economics. Those who were interested enough to read the series of resumés of the Survey in different counties published in the organization section of the *Journal of the American Medical Association* were well repaid for their efforts. Not alone was an inventory given of medical care in the respective counties such as never appeared before, but the reports revealed inadequacies in certain fields and pointed out methods of correction and improvement which should be of great value to the public welfare of the areas involved.

Cook County Appraisal Thorough

An example of thoroughness and intelligent appraisal of the supply of medical care was revealed in the report made in Cook County by the Committee on Medical Economics of the Chicago Medical Society. For thoroughness and intelligent consideration of the problems involved this report stands out as a shining example of what could be accomplished through a Survey made by physicians coöperating with agencies allied in the care of the sick. The difficulties encountered in conducting a survey of this kind in a community like Chicago are self-evident. Too much praise cannot be given to the members of this committee, headed by the well known urologist, Dr. Herman Kretschmer.

System Meets Demand

In the introduction to their report they called attention to the difference, which was suggested by Miss Josephine Roche, between *active demands* and *actual needs* of medical care. The difference between these terms is considerable. The committee's most important and final conclusion was that the supply of medical care in Cook County was sufficient to meet the *active demand* for all urgent requirements. This very important conclusion was arrived at only after a most thorough and painstaking search for facts, made by physicians with the aid of all allied agencies as well. The results of this investigation should help to answer the criticism so frequently made that the present system of distribution of medical care is inadequate to meet the demands of the community.

Important Needs Uncovered

Although this Survey revealed that the supply of medical care was sufficient to meet the active demands of the community, the committee was convinced that many very important needs exist. We quote from their report as follows:

"Our ideal must be to provide complete medical care, including both active demands and actual needs, for all who should have it. To achieve such an ideal means the formulation of measures which will provide medical care for the entire population at some periods in their

lives, including those who seek it actively in addition to those who do not realize, and those who are unable to recognize their own needs for it, and those who will not accept it. *The accomplishment of such a program requires continuous and progressive improvement with respect to professional technics and mental attitudes of all who participate in giving any part of complete medical care. To this end, as the plan develops, the public at large must realize and evaluate the nature of this effort, its difficulties and successes. Thus there is emphasized the importance to all of public relations on a broad and sincere basis and of general education."*

Education Required

They emphasize the fact that lack of medical care which may exist is based on ignorance on the part of the public as to its needs. It has been reliably stated that 10 per cent of the population does not wish medical care. *The greatest need shown by the study is expressed by the one word "education."* Education includes not only that of the medical profession itself, but of all the professions and technical groups having to do with health activities, as well as the education of the general public.

For Profession and Public

A. Education of the professions giving medical care is specifically recommended. The desirability of coöperation with the pharmaceutical profession is referred to. They emphasize one objective, namely the reduction of expense of medication from unwarranted prescription of proprietary preparations.

B. Education of the public particularly, and the appointment of a Committee on Education in regard to Medical Care.

C. Stimulation of college and university faculties to include health education in their curricula.

Nation-wide Program Recommended

The Committee favored the institution of a continuous nation-wide program of education of the general public by a Committee on Education in Regard to Medical Care, with an active membership and a full time secretary. All members of this Committee should have the broadest human interest and sympathy, in addition to exceptional professional qualifications. The profession should promptly undertake devising ways and means of providing individual physicians available to the medically indigent and those in the lower income groups.

For a Coördinated System

Under Hospital Needs, they recommend:

1. That all government hospitals in Cook County except state and federal hospitals be administered by a single metropolitan hospital commission. The committee believes the chaotic hospital system in Chicago and Cook County is the direct result of absence of a coordinated hospital system under a single controlling body.

2. More widespread distribution geographically of facilities, using as far as possible existing hospital and clinical facilities, by government subsidy if necessary.

More Subsidized Beds

3. More subsidized beds for children and maternity cases, and additional beds for colored patients, for patients with active tuberculosis and nervous and mental diseases.

4. More facilities for the care of chronic and incurable patients, not necessarily in hospitals.

They call attention to the persistence of a higher tuberculosis mortality rate for the county and the state of Illinois and the fact that the death rate for this disease has decreased very little during the past six years.

5. All unapproved hospitals to be brought up to standard to secure admission to the list of registered hospitals.

Public Ambulance Service Advocated

6. The Public Ambulance Service Plan should be adopted, which would expedite emergency care. The deplorable lack of an organized emergency ambulance service in the City of Chicago materially enhances the hazards for accidents in this area.

The committee calls attention to the well-known need and present lack of *nursing care* to be provided for the sick in their homes. Particularly is this true of low cost practical nurses. Regulation and control of training of such nurses should be supervised by organized medicine. It would seem that if the government really wishes to be of help to the sick with low

incomes, there would be no more practical way than to develop this type of nursing and subsidize it.

Agency for Needy Patients

The next proposal is of considerable importance, namely that the office of the Chicago Medical Society should include a *Department of Medical Care.* The director of this department should receive reports of persons in immediate need of medical care which they cannot secure and investigations should be made, with a view to adjustment. This department would assist welfare agencies and other organizations about any phase of medical care. To this committee any information about any phase of medical care would be brought, and conditions suggested which might be improved by the action of the Medical Society.

For Supervised School Health

The large field of health services in the schools should have the continuous and considered attention of the medical society, through a standing committee. To them should be given the responsibility of providing progressive leadership in this field, including the public, parent-teacher associations, and the school personnel.

Ironic Contrast

In contrast to this thorough, painstaking appraisal of the health situation and the intelligent recommendations for improvement submitted by the Chicago and many other County Medical Societies, look at the inexact methods employed by the Federal Government in formulating the so-called National Health Program. Based on slip-shod, ridiculous methods of investigation, and on the visionary plans of health theorists, it is moulded in a form so as to have the greatest political and sentimental appeal. It is for such indefinite proposals that hundreds of millions were requested in the Wagner Health Bill, without any exact ideas as to needs. *If the Federal Health authorities really wished to find out where the greatest needs actually exist and the best way to solve the problems involved, they would consult and work with physicians and their organizations, who know. Have they done so? Tragically, and ironically, No!*

ADVANCE NOTICE

Commentators on legislation passed by the 76th Congress are nearly all of one mind on the question of future enactments on health.

They are certain that the 77th Congress will see great expansion of public health services. Political dopesters see health legislation in the platforms of Republicans as well as New Deal Democrats. They foresee that the new Federal Security Chief, Paul V. McNutt, will bend his energies principally to the expansion of Social Security services to include more health service as one sure bid to popular favor.

Senator Robert Taft, while proceeding cautiously so as not to give offense to conservative opinion nevertheless ventured to make the following suggestion at the laying of the corner stone of the new Doctors' Building in Washington.

"Federal Aid Can Be Worked Out"—Taft

"There is hardly a field in which there has been more sensational and continuous improvement than that of medicine in the United States. That improvement has been due to the brilliant, unselfish and industrious work of thousands of physicians. It is not their fault that incomes are unequally distributed and that efforts by local governments to cover the entire field of health have been restricted by lack of resources. *But now I hope they will take an active interest in seeing that the unequalled medical service received by most Americans is extended to the entire population.* Their own interest and participation in the program will make it certain that it is not dominated by half baked theorists or by those who believe in a totalitarian state, directing the lives and caring for the health of all of its citizens through the mechanical and usually careless action of government bureaus. I believe a federal aid program can be worked out. I believe it can be much simpler and much more economical and much more likely to preserve the essential independence of the doctors than the present Wagner bill. I believe it can be worked out with the assistance and coöperation of the doctors themselves."

In a recent issue of the *United States News* the following paragraph appeared:

"The next drive to expand the Social Security system will be to incorporate a public health program. Top social security administrators believe Congressmen can be persuaded to vote liberal funds for expanded health services."

May Need Publicly Supported Facilities—U. S. Public Health Service

In a current statement by the United States Public Health Service issued to mark its removal from the treasury department to the new Federal Security Agency there is the following observation:

. . . "Recent enactments of Congress reflect the changing concepts of the field of public health and the changing emphasis on the various activities—from general quarantine and sanitation measures to the provision of adequate health facilities for all the people—from diseases already so successfully reduced and controlled by modern sanitary and other public health measures, to those heretofore unattacked or unsuccessfully combatted such as venereal diseases, cancer, heart disease and other chronic conditions of the older age group. Not that earlier basic measures are neglected; for in some instances, as in the case of yellow fever, it has recently been found necessary to extend quarantine inspection activities, especially as the result of speedy airplane travel and the existence of foci of jungle fever in South America and the possibility of animal reservoirs and as yet unknown vectors of the disease; but rather greater attention is now being paid to the extension of health facilities and to research in fields which offer greater hope and promise of reduction of disease and suffering. *It is now realized, also, that adequate medical care for large numbers of our population cannot be provided without a greater measure of public subvention.* By reason of an inherent interest in the problem, fundamental organization and medical knowledge, the health organizations, Federal and State, are the best qualified and equipped agencies to take leadership in reducing the amount, severity and economic consequences of illness. *To be effectual, this leadership may, in the future, of necessity include in the public health program, the operation of publicly supported facilities and services in which medical care and other means for the improvement of human health and welfare provide the central purpose;* and provision for this activity in the field of health advancement may be interwoven into the future pattern of public health."

Many Experiments

Meanwhile in many states and counties a variety of plans for payment of medical bills are in process of organization under medical sponsorship. They range from so-called coöperatives to cash indemnity insurance and will in time provide useful figures based upon experience on which to estimate their feasibility and worth.

Until this information is available, however, there is every reason for avoiding any health legislation that will include even a medically sponsored program for health insurance.

Physicians everywhere may congratulate themselves, therefore, that no action was taken at this session of Congress on the Wagner bill S 1620; but their task has just begun.

Friends and foes, alike, must be made aware of the fallacies and futilities that lie ahead of government "subvention" in the actual care of the sick. There will undoubtedly be increased appropriations for health services. It is the responsibility of organized medicine to see that those appropriations are made available where they are needed most and that they do not operate to fasten compulsory health insurance, European plan, upon Americans.

FAMILY DOCTOR—MYTH OR REALITY?

A new and subtle challenge to defenders of the private practice of medicine has just been launched by the redoubtable Mr. Michael M. Davis, chairman of the Committee on Research in Medical Economics, in a recent issue of the *Survey Graphic.*

The chief assets of our American system of medicine are the free choice of physician which it permits and the preservation of a personal relationship between patient and physician.

These two assets were cited by the House of Delegates of the American Medical Association as essentials to good medical service which would be lost under any system of compulsory government medicine.

Basing his observations on a study of 365 New York families just above the relief level, Mr. Davis declares that the realities do not correspond to this official and imaginative description of our American system.

Choice "Haphazard"

In his sample of low income families the family doctor was practically non-existent, Mr. Davis says; there was no personal relationship of any permanent character between the patient and his doctor and, in the course of a year, medical services needed by a family were usually secured in a haphazard and uncoördinated fashion.

Furthermore, he hazards the guess, based chiefly upon personal observation in the course of his studies, that the family in the upper income brackets is likely to have—not one family doctor who in turn guides them to specialists

as needed—but four or five specialists more or less haphazardly selected. Only the children in charge of a pediatrician have anything resembling a coördinated service.

What Do They Know at Vassar?

And as to free choice—he says choice of any kind is made difficult because of the difficulty of getting at lists of qualified physicians. "You can find out if you know the ropes," he says, "but how many graduates from Yale or Vassar would know the professional ropes"?

The answer to this study is obvious. The fact that free choice of physician is not always wisely used and that the personal relationship between patient and doctor does not always operate to its most beneficent extent does not, of course, invalidate the principles of free choice and personal relationship. Neither does it cast doubt upon their importance to Americans.

Education Needed

As a matter of fact, the conditions discussed by Mr. Davis probably exist in the largest cities, only, and scarcely at all in the smaller communities and the rural districts.

On the other hand, better general education in existing aids to an intelligent choice is undoubtedly needed. Such education should also include much greater emphasis on the rôle of the permanent family advisor as the highest development of medical service and the essential factor in coördinated medical service.

NEW PROBLEMS IN RELIEF

There are difficulties ahead for administrators of relief in the next few months and, presumably, also for physicians who take care of relief clients.

The relief appropriation for the biennium, 1939 and 1940, has been reduced and available funds will be still further depleted if any large number of WPA employees are returned to direct relief.

Furthermore, there is a new relief administration at the capital. Following the plan of the Re-Organization Bill, the old SRA was destroyed and a new Division of Social Welfare, part of a new Division of Social Security came into being. The division has a new administrator, Mr. Walter W. Finke, whose assistant is Mr. William H. Crowe. Both are well qualified for the work but new to their jobs. Many experienced staff members are gone and in their places are recent appointees who must learn all the details of their complicated tasks.

Confusion Probable

There is every reason to believe that Mr. Finke and his Department as well as Mr. Carl H. Swanson, new head of the Division of State Institutions, will coöperate closely with the medical profession. But it is quite possible that there will be confusion and misunderstandings before the new division is fully organized and running smoothly.

It is possible, also, that the new administration will not be familiar with medical policies and precedents in Minnesota and will need the same assistance that was given to FERA and SRA heads by the Council in the early days of relief in Minnesota.

Total Mounts

The total number of persons benefitting by relief, WPA employment or Social Security Aids in Minnesota is still appallingly high. The approximate figure is 491,000 and that total does not completely cover Farm Security clients, CCC enrollees, or recipients of NYA aids.

The immediate interest of the physician in this problem concerns provision of essential emergency medical aid for these people. He must see that they get the care they need and also that sound principles of medical practice are safeguarded.

Unique Legislation

Close contact will be maintained with division heads by the Council and the State Office. Individual physicians in their home communities must work closely with county welfare boards, also, in the solution of local problems of relief.

In many respects the Minnesota State Medical Association finds itself confronted with almost the same task that confronted the Council of the association a few years ago.

But there is this important difference: essential legislation giving the relief client his choice of physician is on the statute books, an accomplishment which is unique among most states of the country.

THE NUISANCE SUIT

(Monthly Editorial Prepared by the Medical Advisory Committee)

The American people have become insurance-minded to the highest degree. There are very few either real or anticipated catastrophes that cannot be insured against, and there are very few of the events of life in which insurance benefits cannot play a part.

The fact that indemnity insurance is so prevalent, of course, makes for litigation—litigation based on real or fancied wrongs or injury to property or person.

Your Medical Advisory Committee divides malpractice litigation, in which we are particularly interested, under three general heads:

1. That due to a genuine injury committed by the defendant in the suit, to the person of the plaintiff.

When a real injury has been committed, the suit should be settled at once. There is nothing gained by a long, expensive trial. However, the facts must not be open to question and they must plainly show the defendant guilty of the alleged wrong doing.

2. A fancied wrong committed by the defendant to the body of the plaintiff.

The case of the fancied wrongful act is usually the one brought for large amounts. Here the facts in the case do not show any just cause for action and are based solely on the hopes, not so often of injuring the reputation of the physician involved, as on the wish to obtain money from the insurance company. These cases should be fought to the limit and every recourse in the courts exhausted before any damages are paid.

3. The nuisance suit brought in the hope of obtaining either a small amount of money or nullification of the account owing for professional services.

A nuisance suit is an annoying attempt to vilify the good work of a physician by one who is of unscrupulous character, not only in his dealing with medical men, but in all his dealings in business and commercial life. Indemnifying insurance companies should not too readily compromise these cases. One settled in this way leads to other "trumped up" cases—a vicious circle for which all holders of insurance pay.

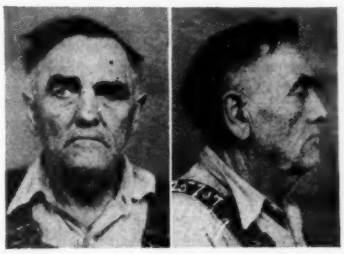
Insurance should in most so-called "malpractice cases" defend, not indemnify.—B.J.B.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

Saint Paul Abortionist Given 10 to 15 Year Prison Sentence

Re. State of Minnesota vs. Peter H. Nellessen.

Peter H. Nellessen, sixty-eight years of age, 1054 Western Avenue, Saint Paul, married and the father of six children, was sentenced to a term of not less than ten and not more than fifteen years at hard labor in the State Prison at Stillwater, on July 25, 1939, by the Honorable Kenneth G. Brill, Judge of the District Court for Ramsey County. Nellessen entered a plea of guilty to an information charging him with the crime of manslaughter in the first degree following the death of a twenty-eight-year-old, married, Saint Paul woman on July 19, 1939. Nellessen had performed a criminal abortion upon the deceased on July 3, 1939, for which he admittedly received the sum of $15.00.



Nellessen served a previous term of three years in the State Prison at Stillwater, following his plea of guilty, in Saint Paul, in July, 1935, to an information charging him with the crime of abortion. Nellessen holds no license to practice any form of healing whatsoever in the State of Minnesota. He stated that about forty years ago he worked for a number of years as a male nurse at the Soldiers' Home at Minnehaha Falls. He gave his occupation at the time of his arrest as a stone mason. According to the records of the Bureau of Identification of the Saint Paul Police Department, Nellessen has six previous convictions on various liquor law charges, the sentences ranging from five months in the county jail, to eighteen months in the Federal Penitentiary at Leavenworth, Kansas.

Spring Valley Woman Pays $100 Fine for Illegal Practice of Healing

Re. State of Minnesota vs. Elizabeth Schulz.

On August 14, 1939, Elizabeth Schulz, forty-seven years of age, entered a plea of guilty before the Honorable Norman E. Peterson, Judge of the District Court, at Preston, Minnesota, to an information charging her with practicing healing without a basic science certificate. Following a statement of the facts to the Court, Judge Peterson sentenced Mrs. Schulz to pay a fine of $100 or to serve thirty days in the county jail of Fillmore County, in the event the fine was not paid. Mrs. Schulz promptly paid the fine.

Mrs. Schulz was arrested following the filing of a complaint against her by Mr. Brist on behalf of the Minnesota State Board of Medical Examiners on August 11, 1939. The investigation made by the Medical Board disclosed that Mrs. Schulz had been seeing numerous patients at her home in Spring Valley where she attempted to diagnose and treat their ailments. Mrs. Schulz would charge each patient 50 cents per call, and in addition to administering a sort of massage treatment, she would write out a prescription on the prescription blanks of the Sward-Kemp Drug Store at Spring Valley, Minnesota, for such drugs and medicines as she thought the patient should have. While Mrs. Schulz did not sign these prescriptions she had an

understanding with Mr. E. B. Petersen, a part owner of the drug store, that they would be honored. Other equipment used by Mrs. Schulz, in her practice, consisted of a stethoscope, numerous dressing forceps and other similar articles.

Mrs. Schulz has a previous conviction for a similar offense in the same court in 1934, and at that time was sentenced to three months in the Fillmore County Jail, but the sentence was suspended and she was placed on probation. At that time Mrs. Schulz was selling a mineral food. On January 10, 1934, one Russell Prinsen, nine years of age, died of diabetes. Russell had been under Mrs. Schulz' care and was taking this mineral food. Mrs. Schulz promised Judge Peterson then that she would refrain from practicing healing in any manner in the future. However, she again resumed the practice which resulted in her arrest in the present case. When Mrs. Schulz was arraigned before Justice Fraser at Preston, Minnesota, on August 12, 1939, she waived her preliminary hearing and was held to the District Court. She indicated a desire to enter a plea of guilty and have the matter disposed of at once, which was done.

It is to be hoped that two prosecutions have convinced Mrs. Schulz that she cannot engage in the practice of healing in any manner in the State of Minnesota. The only training that Mrs. Schulz has ever received along these lines, is a short time as a student nurse some twenty-five years ago, at the State Hospital at Fergus Falls. Mrs. Schulz had in her possession at the time of her arrest, a diploma from the Weltmer Institute of Suggestive Therapeutics at Nevada, Missouri, which was dated July 28, 1933. This diploma purports to confer upon Mrs. Schulz the right to make practical application of Suggestive Therapeutics. She stated that she attended this school in the summer of 1933 for a period of four weeks and that the tuition fee was $50. Mrs. Schulz, and her lawyer, stated to the Court that Mrs. Schulz would absolutely refrain in the future from practicing healing in any way, shape or manner. The Medical Board feels that Mr. Petersen's connivance in the operation of this scheme by Mrs. Schulz, is not to his credit as a registered pharmacist, and is a violation of the laws of this state. Mr. Petersen was also advised that any further violation of the medical laws of this state on his part, will result in the prompt filing of a complaint by this Board.

The State Board of Medical Examiners wishes to acknowledge the friendly and courteous coöperation of Mr. Clarence T. Perkins, County Attorney of Fillmore County, in the prosecution of this case.

PHYSICIANS LICENSED ON JULY 6, 1939

June Examination

Barnett, Joseph Morton, U. of Minn., M.B. 1938, Minneapolis, Minn.

Birnberg, Victor Jack, U. of Minn., M.B. 1938, Minneapolis, Minn.

Blackwell, William Joseph, Northwestern, M.B. 1935; M.D. 1936, Rochester, Minn.

Blomberg, Robert David, U. of Minn., M.B. 1939, Flint, Mich.

Brooke, James William, U. of Ore., M.D. 1938; Lancaster, Minn.

Burks, James Willis, Jr., Wash. U., Mo., M.D. 1937, Rochester, Minn.

Christiansen, Harold Aug., U. of Minn., M.B. 1938; M.D. 1939, Minneapolis, Minn.

Coulter, Everett Benjamin, U of Minn., M.B. 1939, Minneapolis, Minn.

Coventry, Markham Bingham, U. of Mich., M.D. 1937, Rochester, Minn.

Craig, David Mark, U. of Minn., M.B. 1939, Wauwatosa, Wis.

Cronin, Donald Joseph, U. of Minn., M.B. 1939, Minneapolis, Minn.

Douglas, Kenneth Wallace, U. of Minn., M.B. 1939, Tacoma, Wash.

Engstrom, William Weborg, U. of Minn., M.B. 1939, Minneapolis, Minn.

Fink, Daniel Louis, U. of Minn., M.B. 1938, Minneapolis, Minn.

Fogarty, Charles William, Jr., U. of Minn., M.B. 1938, St. Paul, Minn.

Forsythe, James Robert, U. of Minn., M.B. 1939, Brooklyn, N. Y.

Fredricks, Merriam Gerard, U. of Minn., M.B. 1939, Wauwatosa, Wis.

Goehrs, Gilman Henry, U. of Minn., M.B. 1938, St. Cloud, Minn.

Haisten, Arnold Sessions, U. of Pa., M.D. 1936, Rochester, Minn.

Hampton, Hiram Phillip, Emory U., M.D. 1937, Rochester, Minn.

Hay, Lyle Joseph, U. of Minn., M.B. 1937; M.D. 1938, Minneapolis, Minn.

Heckel, Donald Q., U. of Minn., M.B. 1939, San Bernardino, Cal.

Jacobson, Charles Edward, Jr., Cornell U., M.D. 1935, Rochester, Minn.

Jacobson, Wyman E., U. of Minn., M.B. 1939, Flint, Mich.

Kaplan, Harry Arthur, U. of Minn., M.B. 1937, Duluth, Minn.

Kelly, Clarence Andrew, Marquette U., M.D. 1939, Dresser Junction, Wis.

Kelsey, Chauncey Miller, U. of Minn., M.B. 1939, Hinckley, Minn.

Koskela, Lauri Edwin, U. of Minn., M.B. 1938, Coleraine, Minn.

Lindert, Merlyn Carl Fred, U. of Minn., M.B. 1938, Wauwatosa, Wis.

Lindley, Stanley Bryan, U. of Minn., M.B. 1938, Minneapolis, Minn.

MacMillan, David Glenn, U. of Minn., M.B. 1939, Duluth, Minn.

MacPherson, Malcolm Morrison, U. of Manitoba, M.D. 1935, Rochester, Minn.

Magner, Charles Edgar, Rush Med. Col., M.D. 1938, Malta, Mont.

Monserud, Nels Ordell, Rush Med. Col., M.D. 1938, Cloquet, Minn.

Mooney, Robert Davis, U. of Minn., M.B. 1938, St. Paul, Minn.

Morrison, William Forster, U. of Minn., M.B. 1938, Minneapolis, Minn.

Musachio, Nicholas Frank, U. of Minn., M.B. 1938, Eden Valley, Minn.

O'Leary, John Hall, U. of Minn., M.B. 1938, Minneapolis, Minn.

Phalen, George Smith, Northwestern, M.B. 1937; M.D. 1938, Rochester, Minn.

Raszkowski, Harvey Joseph, U. of Wis., M.D. 1936, Rochester, Minn.

Richardson, Robert Joseph, U. of Minn., M.B. 1938, Rushford, Minn.

Satory, John Joseph, U. of Minn., M.B. 1938, Wabasha, Minn.

Schwyzer, Hanns Carl, U. of Minn., M.B. 1938, Saint Paul, Minn.

Selmo, Joseph, Loyola U., M.D. 1939, Caspian, Mich.

Sickels, Edward Worman, U. of Minn., M.B. 1939, Detroit, Mich.

Stewart, Marvin Jerome, U. of Minn., M.B. 1939, St. Paul, Minn.

Stoen, Harold Jennings, Rush Med. Col., M.D. 1936, Cleveland, Ohio.

Swenson, Orrin Endre, U. of Wis., M.D. 1938, Stoughton, Wis.

Trow, James Edward, U. of Minn., M.B. 1939, New Orleans, La.

Veranth, Leonard Anthony, Marquette U., M.D. 1939, Ely, Minn.

Weisman, Sydney Jerald, U. of Minn., M.B. 1939, Buffalo, New York.

Weismann, Rodger E., U. of Iowa, M.D. 1938, Houston, Minn.

Westrup, John Edward, Marquette U., M.D. 1939, Maple Lake, Minn.

Williams, Bill Henry, U. of Minn., M.B. 1937; M.D. 1938; Minneapolis, Minn.

Williams, John Alexander, U. of Minn., M.B. 1939, Duluth, Minn.

Zinter, Ferdinand A., U. of Minn., M.B. 1938, Minneapolis, Minn.

By Reciprocity

Clark, Richardson Evans, U. of Iowa, M.D. 1936, Spring Valley, Minn.

Eyres, Thomas Edward, U. of Iowa, M.D. 1932, Pequot, Minn.

Pewters, John Thomas, U. of Minn., M.B. 1937; M.D., 1938, Minneapolis, Minn.

Witherspoon, Jackson Thornwell, Johns Hopkins U., M.D. 1928, Minneapolis, Minn.

National Board Credentials

Cunningham, Bernard Poland, New York Univ., M.D. 1936, Rochester, Minn.

Olson, Lorin Magnus, Northwestern, M.B. 1938; M.D. 1939, Chisago City, Minn.

◆ REPORTS and ANNOUNCEMENTS ◆

MEDICAL BROADCAST FOR SEPTEMBER

The Minnesota State Medical Association Morning Health Service

The Minnesota State Medical Association broadcasts weekly at 11:00 o'clock every Saturday morning over Station WCCO, Minneapolis (810 kilocycles or 370.2 meters) and Station WLB, University of Minnesota (760 kilocycles or 395 meters).

Speaker: William A. O'Brien, M.D., Associate Professor of Pathology and Preventive Medicine, Medical School, University of Minnesota. The program for the month will be as follows:

September 2—Cause of Accidents
September 9—First Aid
September 16—Accidental Infection
September 23—Shock and Hemorrhage
September 30—Injuries of the Teeth and Jaws

INTERNATIONAL ASSEMBLY OF THE INTER-STATE POSTGRADUATE MEDICAL ASSOCIATION

This year's International Assembly of the Inter-state Postgraduate Medical Association of North America will be held in the Palmer House, Chicago, October 30 and 31 and November 1, 2 and 3.

This assembly has been for a number of years one of the outstanding medical meetings of national and even international scope. A full program of clinics and addresses has been arranged for morning, afternoon and evening of the five days of the assembly. The dinner scheduled for Wednesday evening will be addressed by Major General James C. Magee, surgeon-general of the United States Army, and Major General Ross T. McIntire, surgeon-general of the United States Navy, with Dr. George W. Crile, president of the assembly, master of ceremonies.

Professor Dott. Emanuele Momigliano of the Department of Obstetrics and Gynecology of the Royal University of Rome and Dr. Alva H. Gordon and Dr. John R. Fraser of McGill University constitute the visitors from beyond the bounds of the United States.

Outstanding teachers and clinicians from medical centers throughout the country will provide a full program of interest and practical value, not only to the specialist, but also to the general practitioner. Those who have attended one of these meetings know the high caliber of the programs.

The Saturdays preceding and following the assembly will be devoted to clinics in the Chicago hospitals. Chicago's hospitals, hotels and central location make it particularly suitable for a large meeting of this kind. All physicians in good standing with their state or provincial societies are cordially invited to attend. A nominal registration fee of $5.00 admits to all the scientific meetings.

Members of the profession are urged to bring their ladies as an excellent program is being arranged for their benefit by the Ladies' Committee.

A program is being mailed to all physicians in good standing in the United States and Canada.

On page xix of the advertising section of this number of the JOURNAL appears a list of the teachers and clinicians who are to take part in the program of the assembly.

Dr. George W. Crile is president and chairman of the program committee this year and Dr. Chevalier Jackson is president-elect. Dr. William B. Peck, Freeport, Illinois, is managing director.

GRADUATE FORTNIGHT—NEW YORK ACADEMY OF MEDICINE

The twelfth annual Graduate Fortnight of the New York Academy of Medicine, this year devoted to the subject of the Endocrine Glands and Their Disorders, will be held October 23 to November 3, 1939.

Sessions will consist of morning Round Table Conferences and evening lectures at the Academy headquarters and afternoon clinics at various hospitals. The program contains the names of outstanding authorities in this country and Canada on Endocrinology.

Copies of the program and further information may be obtained by writing the New York Academy of Medicine, 2 East 103rd Street, New York City.

1940 STATE MEETING

The next annual meeting of the Minnesota State Medical Association will be held in Rochester, Monday, Tuesday and Wednesday, April 22, 23 and 24, 1940. The Council and House of Delegates will meet on Sunday, April 21. Scientific sessions will be held in the new Rochester Auditorium. Dr. F. J. Heck, Rochester, is chairman of local arrangements.

In Memoriam

Benjamin W. Kelly

1874-1939

DR. B. W. KELLY died very suddenly at his home in Aitkin, Minnesota, June 4, 1939, at the age of sixty-five. He had suffered from poor health since February when he sold his practice to Dr. I. L. Mitby on retiring.

Dr. Kelly was born in Tawas City, Michigan, in 1874. After working his way through the medical school of the University of Michigan and graduating in 1897, Dr. Kelly established an office at West Farmington, Ohio, where he remained for a year. In 1898 he moved to Aitkin, Minnesota, where he practiced until his retirement this year.

The hardships encountered in the early years of his practice, with poor roads, doubtless accounted for the interest he took in road improvement. At the time of his death he was president of the Northwest Good Roads Association and he had been president of the Aitkin and Minnesota Automobile Associations.

At the time of the World War, Dr. Kelly enlisted as a lieutenant in the Medical Corps. He was first put in charge of an infirmary at Hancock, Georgia, and later was surgeon in charge of troops on the *S.S. President Lincoln* on the ship's last trip before it sank. He served as inspector at Camp St. Magaire and was regimental surgeon in the first air service at Les Vasges. Later he was surgeon for the Epinol district and before his return to the United States had achieved the rank of lieutenant colonel. In 1924 he was made colonel of the Reserve Corps. He was a past commander of the Aitkin American Legion Post and a member of the Veterans of Foreign Wars.

Dr. Kelly was one of the first health officers of Aitkin County. He was past president of the Upper Mississippi Valley Medical Society and a member of the Minnesota State and American Medical Associations. He also held membership in the Association of Military Surgeons of the United States.

Dr. Kelly was a charter member of the Aitkin Lions Club. He was also a member of the Minnesota Safety Council and of the Minnesota Law and Order League. He had served as director of the Minnesota State Sanitary Conference. A 32nd Scottish Rite Mason, Dr. Kelly was a pastmaster of the Aitkin lodge No. 213 A.F. and A.M.

While medicine was Dr. Kelly's main interest, his next thoughts were for his community and state, working especially for road improvement. He was a great lover of nature and was very fond of Mesquaghughik, his country place at Cedar Lake near Aitkin.

James Alfred Watson

1867-1939

DR. JAMES ALFRED WATSON was born in Longford, Ireland, on September 5, 1867, and died in Minneapolis, June 17, 1939.

Dr. Watson came to Canada at the age of seventeen with his father. They were followed shortly by his mother and three brothers, and the family settled on a farm at Pilot Mound, in Manitoba. By means of teaching in the elementary schools, he was able to finance his medical education at the University of Manitoba, in Winnipeg, graduating in 1894. He then went to Wood Lake, Minnesota, to engage in the practice of medicine. In 1898 he left Wood Lake to undertake postgraduate study in eye, ear, nose, and throat work in New York and Vienna, returning to Minnesota in 1900. In this year he married Lucia Coghlan of Wood Lake. He then commenced the practice of opthalmology and otolaryngology in Minneapolis, and continued in this actively until June 15, 1939, just two days prior to his death.

During the earlier years of his practice in Minneapolis, he was a member of the faculty of Hamline University Medical School.

Since 1920 he had practiced as a member of the Eye, Ear, Nose, and Throat Clinic, in close association with Drs. Eugene Strout, John Macnie, and W. E. Patterson. Until 1932, the clinic was housed in their own building at 74 South 11th Street, after that time in offices in the Medical Arts Building.

Dr. Watson became a citizen of the United States not long after entering practice in Minneapolis. During the entire period of his life in Minneapolis he was an active member of Hennepin Avenue Methodist Episcopal Church.

He was a member of the following associations and clubs: Hennepin County Medical Society, Minnesota State Medical Association, American Medical Association, American College of Surgeons, American and Minnesota Academies of Opthalmology and Otolaryngology; Minneapolis Athletic Club, Lafayette Club.

Surviving are his wife and four children, Cecil James, Leland Alfred, Elinor Isabel, and James Alfred, Jr., all of Minneapolis.

During the long period of his practice and life in Minneapolis, countless associates, patients, and acquaintances had known him as a sound and conservative practitioner of medicine, and as a true and kindly friend.

OF GENERAL INTEREST

Dr. D. W. Cummings, formerly of Waseca, is now located in Byron, Minn., where he opened offices August 1.

* * *

Dr. Oscar Lipschultz announces the opening of his office for the practice of x-ray diagnosis and therapy at 506 Medical Arts Building, Minneapolis, Minnesota.

* * *

Dr. J. W. Erickson of Minneapolis opened offices for the practice of medicine in Jackson the first of August. Dr. Erickson recently completed a year's interne work at Swedish Hospital.

* * *

Dr. R. W. Campbell and Dr. C. H. Coombs of Cass Lake have combined their respective offices and will maintain headquarters in the recently incorporated Cass Lake General Hospital.

* * *

Dr. J. W. Brooks, for the past year member of the medical staff of the General Hospital, Madison, Wis., has located at Lancaster and has established his residence there. He is associated with Dr. J. J. Stratte and Dr. A. S. Berlin of Hallock.

* * *

Dr. E. N. Nelson has opened a medical office in Lake Park. He comes from Cashton, Wis., and is a graduate of the University of Cincinnati, class of 1938. He interned at the Cincinnati General Hospital and the Swedish Hospital in Minneapolis.

* * *

Dr. Manford B. Dahle, a graduate of Rush Medical College, has opened offices for the practice of medicine at Warroad, Minnesota. Dr. Dahle plans to devote a good share of his time to surgical work, besides carrying on a general practice.

* * *

Dr. Robert W. Merrill, who was associated with the late Dr. J. F. Cumming in the practice of medicine in Morris, has bought the practice and equipment of his late associate. Dr. Merrill graduated from the College of Medicine at the University of Minnesota in 1937. Prior to coming to Morris he spent two years in hospital work in Milwaukee, Wis.

* * *

Dr. David P. Anderson, Jr., of Philadelphia, has become associated with the Austin Clinic and will be in charge of surgery. Dr. Anderson is a graduate of the Medical School of the University of Pennsylvania. The past five years have been spent at the Philadelphia General Hospital, the past three as surgical resident. Dr. and Mrs. Anderson moved to Austin, August 15.

* * *

The report has been received that a young man, about twenty-nine years of age, slender and dark complexioned, about five feet eight inches tall, has been visiting doctors in Hibbing and vicinity in quest of morphine for severe renal colic. The presence of blood and pus in the urine offer some grounds for the dispensing of an opiate, but check of his name and residence proved his statements false. These facts are presented as a warning to physicians.

* * *

The Honorable Harlan J. Bushfield, Governor of South Dakota, has appointed a Basic Science Board as authorized by the 1939 South Dakota Legislature. The members are as follows: J. D. Alway, M.D., Aberdeen, South Dakota; F. E. Burkholder, D.O., Sioux Falls, South Dakota; M. L. Severence, Chiropractor, Aberdeen, South Dakota; Wm. H. Waller, Ph.D., Professor of Anatomy, University of South Dakota, Vermillion, South Dakota; Gregg M. Evans, Professor of Chemistry and Physics, Yankton College, Yankton, South Dakota.

* * *

Members of the Minnesota-Dakota Orthopedic Club will serve again this year as clinicians at the fall field clinics conducted by the Bureau of Services for Crippled Children of the State Social Security Board. The clinic schedule has been announced as follows:

Worthington, September 9; Hibbing, September 16; Marshall, September 23; Winona, September 30; Bemidji, October 7; Fergus Falls, October 28; and Mankato, November 18.

Coöperating with the bureau is the Minnesota Public Health Association, the Division of Rehabilitation of the State Department of Education, and Gillette State Hospital.

A letter of referral from the attending physician of the patient is all that is required for entrance to the clinic. All physically handicapped children under twenty-one years of age whose parents cannot provide the needed care are eligible.

* * *

Effective July 1, 1939 applicants for marriage licenses in New York State are now requested to present certification by a licensed physician, not necessarily in New York State, as to the absence of syphilitic disease as shown by physical examination and serological test. The blood test must be performed by a New York City or State Health Department laboratory. Information concerning approved out-of-state laboratories may be obtained from the New York City Department of Health, Worth and Centre Streets, New York City. Blood specimens may be sent by air mail to the Division of Laboratories and Research, New York State Department of Health, New Scotland Avenue, Albany, New York, where examination will be made free of charge.

Further information relative to the marriage of persons in New York State, exclusive of New York City, may be obtained from the Division of syphilis control, New York Department of Health, Albany, New York.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

FUNCTIONAL DISORDERS OF THE FOOT. Their Diagnosis and Treatment. Frank D. Dickson, M.D., F.A.C.S., Orthopedic Surgeon, St. Luke's, Kansas City General and Wheatley Hospitals of Kansas City, Missouri, and Providence Hospital, Kansas City, Kansas; and Rex L. Diveley, A.B., M.D., F.A.C.S., Orthopedic Surgeon, St. Luke's, Kansas City General, Research and Wheatley Hospitals, Kansas City, Missouri, and Providence Hospital, Kansas City, Kansas. 305 pages. Illus. Price, $5.00, cloth. Philadelphia: J. B. Lippincott Co., 1939.

DO YOU WANT TO BECOME A DOCTOR? Morris Fishbein, M.D. 176 pages. Price, $1.50, cloth. New York: Frederick A. Stokes Co., 1939.

CARDIOVASCULAR DISEASES. Their Diagnosis and Treatment. David Scherf, M.D., Associate Professor of Clinical Medicine, New York Medical College, and Linn J. Boyd, M.D., F.A.C.P., Professor of Medicine, New York Medical College. 458 pages. Price, $6.25, cloth. St. Louis: C. V. Mosby Co., 1939.

THE INFANT AND CHILD IN HEALTH AND DISEASE. With Special Reference to Nursing Care. Second Edition. John Zahorsky, A.B., M.D., F.A.C.P. Professor of Pediatrics and Director of Department of Pediatrics, St. Louis University School of Medicine, and Pediatrician-in-chief St. Mary's Group of Hospitals, etc.; and Elizabeth Noyes, R.N., Superintendent and Instructor of Pediatrics, Children's Hospital, San Francisco, California. 496 pages. Illus. Price, $3.00, cloth. St. Louis: C. V. Mosby Co., 1939.

OPERATIVE ORTHOPEDICS. Willis C. Campbell, M.D. 1154 pages. Illus. Price, $12.50, cloth. St. Louis: C. V. Mosby Co., 1939.

TRANSACTIONS OF THE THIRD INTERNATIONAL GOITER CONFERENCE and the AMERICAN ASSOCIATION FOR THE STUDY OF GOITER. 547 pages. Illus. Price, $6.00, cloth. Portland, Ore.: J. C. Hamilton, 1939.

EPIDEMIC ENCEPHALITIS. Etiology, Epidemiology, Treatment. Third Report by the Matheson Commission, Willard C. Rappleye, Chairman. 493 pages. Illus. Price, $3.00, flexible binding. New York: Columbia University Press, 1939.

MANUAL OF THE DISEASES OF THE EYE. Charles H. May, M.D. 16th ed. rev. 515 pages. Illus. Price, $4.00. Baltimore: Wm. Wood & Co., 1939.

This manual, in its fifteenth edition since first published in 1900, is so well known that a review of this last up-to-date publication seems superfluous. It adequately fills the need for which it is intended—a textbook of ophthalmic knowledge for the undergraduate student and the general practitioner of medicine.

C. E. STANFORD, M.D.

OTOLARYNGOLOGY IN GENERAL PRACTICE. Lyman G. Richards, M.D. 352 pages. Illus. Price, $6.00. New York: The MacMillan Company, 1939.

The aim of this book, as frankly stated by the author, is to help the general practitioner determine which cases he should refer to the specialist. It is well written and is a safe and honest presentation of the common diseases of the ear, nose, and throat.

Fifty pages are devoted to the discussion of tonsillectomy and adenoidectomy. The conclusion is that the author considers this operation to be in the field of the general practitioner.

Sinus disease is clearly described, although no mention is made of its treatment by means of suction, either direct or by the Proetz method. The use of hot nasal irrigations is advised. The use of silver protein packs is not recommended. A more complete discussion of sinusitis in children would be expected, in view of the author's extensive experience.

Laryngeal diseases as well as those belonging to the field of endoscopy are carefully described.

The book offers nothing new, but nevertheless can be recommended to the general practitioner as a safe and up to date guide in the diagnosis and treatment of the common diseases of the ear, nose, and throat.

KENNETH A. PHELPS, M.D.

A TEXTBOOK OF NEURO-RADIOLOGY. Cecil P. G. Wakeley, R.Sc., F.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S. (Hon.) Senior Surgeon, King's College Hospital and West End Hospital for Nervous Diseases, et cetera, and Alexander Orley, M.D., D.M. R.E., Fellow of the British Association of Radiologists; Radiologist, West End Hospital for Nervous Diseases, et cetera. 336 pages. 229 illustrations. Price, cloth, $7.25. Baltimore: The Williams and Wilkins Company, 1939.

The authors state that because of the intimate connection between cranial surgery and radiology this work has been undertaken in collaboration between a surgeon and a radiologist. They emphasize the fact that neurology and neuro-surgery have made great advances in recent years and radiology has played its part in this connection. Thanks to the refinement of the radiographic technic and of the radiological interpretation more information can be obtained now from the plain radiograph than was possible a few years ago, while at the same time ventriculography, encephalography and myelography have opened up new fields in the diagnosis of cerebral and spinal lesions and cerebral angiography has proved very valuable for the diagnosis of cerebral pathological conditions, particularly aneurisms. Out of all this has grown a considerable literature, in papers and monographs scattered over a number of countries and this book is an attempt to gather together and correlate all of this, in condensed form, in one volume.

It is an excellent piece of work, giving in compact form a very practical summary of the entire field set forth above. While many of the illustrations are from other sources they have been carefully selected and there are many excellent original diagrams. The book deals well with traumatic lesions of the skull and spine and should have a wide field of usefulness.

GILBERT COTTAM.

LIFE AND LETTERS OF DR. WILLIAM BEAUMONT. Jesse S. Myer, A.B., M.D. Late Associate in Medicine in Washington University, St. Louis. With an Introduction by Sir William Osler, Bt., M.D., F.R.S. Late Regius Professor of Medicine in Oxford University, England. 327 pages. Illus. Price, $5.00. One-half cloth. St. Louis, The C. V. Mosby Company, 1939.

This is a reprint of the volume published twenty-five years ago, with certain additions upon which we shall comment later in this review.

In securing the material for this book, Dr. Myer had access to a unique mass of source material, which he utilized to the full. It seems that throughout his active years Dr. Beaumont had formed the habit of preserving all cogent scraps of information which came into his possession. Letters, memoranda, documents, manuscripts, diaries, clippings, books, et cetera were carefully preserved and after his death they were assembled in two old chests which ultimately found their way into the possession of Mrs. Sarah Beaumont Keim, his daughter, who lived in St. Louis, where Dr. Beaumont had spent the last thirteen years of his life in general practice and where he died in 1853 and was buried in Bellefontaine Cemetery. Mrs. Keim turned over these chests to Dr. Myer and the contents were also examined by Dr. Osler and commented on in his introduction to the facsimile of Dr. Beaumont's original monograph entitled Experiments and Observations on the Gastric Juice and the Physiology of Digestion which was reproduced by the Harvard University Press in 1929, the original book having appeared in 1833.

With untiring zeal and unlimited patience, Dr. Myer went through this motley collection of nondescript material, sorted it out in chronological order as best he could and set aside the items which he felt would best serve his purpose. The result is a remarkably human portrayal of the man as he was and with Dr. Myer's interpolations and comments a sequence is preserved throughout which makes the book read like a fascinating novel, or better, a living biography. It is regrettable that Dr. Myer, then a youngish internist in his prime, should have died shortly after the production of this work in 1912. At least he left a monument that will tend to perpetuate his memory far better than any bronze tablet or even a heroic statue could have done.

The original edition became exhausted and with the untimely passing of its brilliant author so soon afterwards, nothing further was done about it until this year, when Arno B. Luckhardt, writing in the May issue of the *Bulletin of the History of Medicine*, published by the Johns Hopkins Press, about the Dr. William Beaumont Collection of the University of Chicago, had the following to say: "It is hoped that the various libraries, societies, clubs, and individuals will list similarly their Beaumont memorabilia so that a *complete* list of the known Beaumontiana can be published soon in a truly 'Fultonian' manner. If this is done, some future medical historian, with full knowledge of the location and nature of the new source material, should be able to issue a more comprehensive biography and scientific

evaluation of Beaumont than has yet appeared. *Or,* (and here the italics are ours) *the material could be incorporated in a new edition of the scholarly volume of Jesse Myer* (Life and Letters of Dr. William Beaumont, C. V. Mosby Company, St. Louis, Mo., 1912, pp. 317), *still the standard work but hardly obtainable,* as a book dealer ascertained lately when he failed to obtain a copy for which he was willing to pay $75.00. *We certainly owe this effort to America's ranking pioneer physician, gastroenterologist, and model scientific investigator, Dr. William Beaumont."*

Regrettably this excellent advice was too late to reach the publishers, for the present reprint was already in the press, if not actually in distribution. It contains intact the original material written by Dr. Myer, with the correction and verification of certain dates by W. Scott Hancock, and the adequate introduction by William Osler. This would have been quite enough, short of an exhaustive undertaking such as Luckhardt proposed, but the publishers did neither. In their anxiety to adorn the lily they have made three additions which leave this reviewer in the same emotional state as if he had seen the Mona Lisa decorated with lipstick or had listened to the Mendelssohn Concerto in E minor with the cadenza furnished by Irving Berlin. First there is a five-and-a-half page "present-day appreciation" by A. C. Ivy which is both tautological and superfluous, written perhaps to sell the book to those of us who are underprivileged in modern physiology. Osler said it all in one-half the space and his words are as timely today as in 1912. His one paragraph about Prout and Pavlow are enough. Then there is a reproduction in colors of Dean Cornwell's lurid depiction of Beaumont in full dress army uniform, gold lace and all, collecting a specimen of gastric juice from the stomach of Alexis St. Martin in a squalid hut such as "that old fistulous Alexis" undoubtedly occupied. Beaumont's army pay at that time was $40.00 per month, with two to four daily rations, but perhaps the picture is allegorical, a term used by artists to cover the most blatant incongruities. Finally, and here the thing gets ridiculous, are reproduced "Four Letters of Alexis St. Martin," who never wrote a line in his life. They were all written for him, of course, as were all his letters previously, which he used to sign by making his mark, but in the case of these four letters, written the year before his death at about the age of 80, he probably felt too weak and feeble to do that much and allowed whoever wrote the letters to sign his name for him. There is not a line in the book to indicate that these letters were not the work of St. Martin himself. On the contrary, the publishers felicitate themselves in their announcement on page XXXI on securing "several hitherto unpublished letters written by Alexis St. Martin. These will be found in the Appendix." Poor old Alexis, illiterate, besotted, temperamental and a sore trial to his benefactor, had only two things in life: his fistula and his ability to perpetuate his kind. He was the father of seventeen children, four of whom were living at the time of his death.

GILBERT COTTAM

A TEXTBOOK OF OBSTETRICS, WITH SPECIAL REFERENCE TO NURSING CARE. Charles B. Reed, M.D., F.A.C.S., and Bess J. Cooley, R.N. 476 pages. Illus. Price, $3.00. St. Louis: The C. V. Mosby Co., 1939.

This book is offered as a text for student and graduate nurses. The material is presented in condensed form and covers the field of obstetrics in an orthodox manner. The style is more adapted to the medical student than to the nurse.

In the discussion of Anesthesia in Obstetrics, Sir James Y. Simpson is placed in Glasgow instead of Edinburgh. The illustration on page 329 taken from Titus' "Management of Obstetrical Difficulties" is placed upside down.

The book offers little in style or form to compete with a number of excellent texts already on the market.

ARCHIBALD L. MCDONALD, M.D., F.A.C.S.

CORRESPONDENCE

To the Doctors of Minnesota—
A Word of Appreciation

For the past twenty years, eighteen of which have been spent in Minnesota, I have represented the W. F. Prior Company of Hagerstown, Maryland. During this period I have met most of the members of the Minnesota State Medical Association.

It now becomes expedient for me to return to my native Australia for an extended trip, with the probability of remaining there, and I want this brief note to convey to you my deep appreciation for the friendly reception and loyal support and help which you have given me in my business during all these years.

To the several of you who have rendered professional service and friendly counsel I especially tender my sincere thanks.

If any of my many friends and acquaintances among you should ever plan a visit to Sydney I suggest that you write me in advance to 26 Brown Street, Waverley, Sydney, Australia, and while I can't offer you the "keys to the city," I do promise to try to make your stay in Sydney as enjoyable as I possibly can.

My work among you has been pleasant, inspirational and enlightening, as well as profitable materially, and it is with sincere regret that I leave the finest group of people with whom a man could wish to work.

With sincere good wishes for health, happiness and success to all of you, I am

Very sincerely yours,

H. R. (BILL) LAMBERT,
2554 Garfield Avenue South,
Minneapolis, Minnesota

P. S. We are leaving Minneapolis September 10.